

Fourth Edition



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BLACKWELL'S FIVE-MINUTE VETERINARY CONSULT: CANINE AND FELINE



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SPONTANEOUS ABORTION AND PREGNANCY LOSS IN CATS



BASICS

OVERVIEW

- “Abortion” is the delivery of one or more fetuses before it is (they are) capable of surviving outside of the uterus
- “Pregnancy loss” is the death of the embryo, reabsorption of early fetuses, mummification (shriveling or drying up of the fetus, like a mummy), abortion, stillbirths, and outcomes of difficult birth (known as “dystocia”)
- The “queen” is a female cat

GENETICS

- Nonspecific; inbred lines experience higher levels of pregnancy failure than seen in other cats

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Breed Predispositions

- Persians and Himalayans—difficult birth (dystocia)

Mean Age and Range

- Noninfectious causes—more common at the birth following the first pregnancy and in queens greater than 6 years of age
- Infectious causes—all ages

Predominant Sex

- Females

SIGNS/OBSERVED CHANGES in the ANIMAL

- May have no clinical signs, especially early in pregnancy or gestation
- Failure to litter on time
- Decrease in abdominal size
- Weight loss
- Delivery of recognizable fetuses or placental tissue
- Lack of appetite (known as “anorexia”)
- Vomiting, diarrhea
- Behavioral changes
- Discharge from the vulva that contains blood or pus—frequently unnoticed in fastidious queens (that is, queens that groom or clean themselves with meticulous attention) or with early pregnancy or gestational losses; the “vulva” is the external genitalia of females
- Disappearance of fetuses previously documented by physical examination (palpation), ultrasound examination, or X-rays
- Abdominal straining, discomfort
- Depression
- Dehydration
- Fever

CAUSES

Infectious Disease

- Viruses—feline panleukopenia virus; feline herpesvirus; feline calicivirus; feline leukemia virus (FeLV); feline immunodeficiency virus (FIV)
- Bacteria—*Escherichia coli*; *Streptococcus*; *Staphylococcus*; *Salmonella*; *Mycoplasma*; *Mycobacterium*
- *Coxiella burnetii* (Q fever)
- *Toxoplasma gondii* (probably uncommon)

Noninfectious—Reproductive Causes

- Early loss of the embryo
- Difficult birth (dystocia)
- Disease of the lining of the uterus (known as “endometrial disease”)—cystic endometrial hyperplasia (CEH), a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts; very common
- Hormonal disorders
- Inadequate placenta
- Fetal defects—genetic or developmental (anatomic, metabolic, and chromosomal abnormalities)
- Excessive or poorly planned inbreeding and/or poor choices of breeding stock—indirect evidence; difficult diagnoses without thorough family history and test matings
- Effects of social hierarchy in group setting; behavioral disorders in individuals
- Agents that induce abortion (known as “abortifacient drugs”)—luteolytics (such as the prostaglandin, $\text{PGF}_{2\alpha}$); estrogens; prolactin inhibitors (such as cabergoline); steroids; tamoxifen citrate (dogs); mifepristone (dogs); epostane (dogs)

Noninfectious—Nonreproductive Causes

- Nutrition—low taurine intake (taurine is an amino acid [protein] that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain health); nutritional fads; some nutraceuticals
- Severe stress—environmental; physiological; psychosocial
- Trauma
- Some medications
- Consequence of severe, generalized (systemic) disease involving systems other than the reproductive system

RISK FACTORS

- Prior history of pregnancy loss or poor reproductive performance (may be history of individual queen or the cattery)
- Coexistent, severe sudden (acute) or long-term (chronic) disease
- Excessive inbreeding
- Birth following the first pregnancy or queen greater than 6 years of age
- Environmental stress—crowding, poor sanitation, noise, temperature or humidity extremes
- Social order in cattery
- Exotic breeds—often reproduce poorly in large catteries or high-density environments
- Obesity or inappropriate nutrition



TREATMENT

HEALTH CARE

- Outpatient medical management—medically stable patients; suspected hormonal disorders; disease of the lining of the uterus (endometrial disease); noninfectious/nonreproductive pregnancy loss
- Inpatient medical management—abortion imminent or taking place; clinical illness; potential zoonoses—diseases that can be passed from animals to people (unless safe and effective outpatient treatment can be assured); patients being treated with the prostaglandin, $\text{PGF}_{2\alpha}$
- Aborted fetus or discharge may be infectious; isolate patient
- Practice strict sanitation for inpatient or outpatient treatment
- Correct dehydration—administer fluids (such as Normosol® or lactated Ringer's solution)

ACTIVITY

- No limitations, unless an infectious agent is suspected or documented for outpatients
- Isolate infectious patients (preferred)

DIET

- No special dietary considerations for uncomplicated cases
- Persistent diarrhea or other causes of fluid loss—veterinary diet and fluid therapy

SURGERY

- Surgical management—spay or ovariectomy (surgical removal of the ovaries and uterus) for stable patients with no breeding value or if necessary to preserve queen's life



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Depend on underlying causes
- Antibiotics—amoxicillin, pending results of bacterial culture and sensitivity testing
- Prostaglandin ($\text{PGF}_{2\alpha}$)—may be used to stimulate and evacuate the uterus in cases with non-viable fetuses or significant uterine contents noted on ultrasound examination; discuss risks and benefits of prostaglandin treatment with your cat's veterinarian
- Progesterone/progestogens—safe and effective doses for pregnancy maintenance not established; may cause or worsen cystic endometrial hyperplasia (CEH), a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts



FOLLOW-UP CARE

PATIENT MONITORING

- Re-evaluate 7 to 14 days after completion of prostaglandin ($\text{PGF}_{2\alpha}$) treatment
- Repeat ultrasound examination—evaluate uterine evacuation or fetal viability

PREVENTIONS AND AVOIDANCE

- Genetic problems require attention to breeding program
- Infectious causes require surveillance and control measures
- Spay or ovariectomy for cats with no breeding value

POSSIBLE COMPLICATIONS

- Generalized bacterial infection (known as “sepsis”)
- Shock
- Uterine rupture
- Inflammation of the lining of the abdomen (known as “peritonitis”)
- Inflammation of the lining of the uterus (known as “metritis”)
- Inflammation with accumulation of pus in the uterus (known as “pyometra”)
- Bleeding
- Infertility
- Obesity following spay or ovariectomy in mid-life queens
- Cystic endometrial hyperplasia (CEH, a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts) following progestogen therapy; progestogen is any substance capable of producing the effects of the female hormone, progesterone

EXPECTED COURSE AND PROGNOSIS

- Symptomatic retrovirus infection—poor prognosis
- Long-term (chronic) infertility—common after 6 years of age
- Queens not bred prior to 3 to 4 years of age experience higher infertility
- Severe cystic endometrial hyperplasia (CEH, a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts)—recovery of fertility unlikely; inflammation with accumulation of pus in the uterus (pyometra) is a common complication
- Genetic abnormalities causing difficult birth (dystocia) or loss of most or all of litter—guarded to poor prognosis for further reproduction
- Repeated difficult births (dystocias)—recurrence depends on cause; guarded prognosis if cause not ascertained
- Hormonal disorders—often manageable; consider genetic aspects



KEY POINTS

- Zoonoses (diseases that can be passed from animals to people) can be causes of abortion or pregnancy loss in cats; discuss the potential of a zoonosis causing your cat's abortion or pregnancy loss with your pet's veterinarian
- Maintain careful records of reproductive performance for each queen and for the cattery
- Establish disease surveillance and control measures; may require significant changes in cattery management and breeding stock selection
- For breeding cats—consider risks and possible side effects associated with non-surgical solutions, particularly with infectious or genetic causes of pregnancy loss
- Infertility—may result despite successful treatment; may be secondary to conditions pre-existing the pregnancy loss
- Prostaglandin treatment—consider risks and possible side-effects
- Spay or ovariectomy—indicated for primary disease of the uterus for queens with no breeding value

SPONTANEOUS ABORTION AND PREGNANCY LOSS IN DOGS



BASICS

OVERVIEW

- “Abortion” is the delivery of one or more fetuses before it is (they are) capable of surviving outside of the uterus
- “Pregnancy loss” is the death of the embryo, reabsorption of early fetuses, mummification (shriveling or drying up of the fetus, like a mummy), abortion, stillbirths, and outcomes of difficult birth (known as “dystocia”)
- The “bitch” is a female dog

GENETICS

- No genetic basis for most causes of abortion
- Low levels of thyroid hormone due to infiltration of lymphocytes into the thyroid gland resulting in destruction of thyroid tissue (known as “lymphocytic hypothyroidism”); lymphocytes are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process—single-gene recessive trait in borzois

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Familial (runs in certain families or lines of animals) low levels of thyroid hormone due to infiltration of lymphocytes into the thyroid gland resulting in destruction of thyroid tissue (lymphocytic hypothyroidism) reported in borzois—prolonged interval between “heat” or “estrous” cycles, poor conception rates, abortion mid-pregnancy or gestation, stillbirths
- Many breeds considered at risk for familial (runs in certain families or lines of animals) low levels of thyroid hormone (known as “hypothyroidism”)

Mean Age and Range

- Infectious causes, medications causing abortion, fetal defects—seen in all ages
- Cystic endometrial hyperplasia (CEH), a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts—bitch usually is greater than 6 years of age

Predominant Sex

- Females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Failure to deliver or whelp on time
- Delivery of recognizable fetuses or placental tissues
- Decrease in abdominal size; weight loss
- Lack of appetite (known as “anorexia”)
- Vomiting, diarrhea
- Behavioral changes
- Discharge from the vulva that contains blood or pus; the “vulva” is the external genitalia of females
- Disappearance of fetuses previously documented by physical examination (palpation), ultrasound examination, or X-rays
- Abdominal straining, discomfort
- Depression
- Dehydration
- Fever in some patients

CAUSES

Infectious Disease

- *Brucella canis*—bacteria that causes reproductive problems in female and male dogs; disease called “brucellosis”
- Canine herpesvirus
- *Toxoplasma gondii*, *Neospora caninum*
- *Mycoplasma* and *Ureaplasma*
- Miscellaneous bacteria—*E. coli*, *Streptococcus*, *Campylobacter*, *Salmonella*
- Miscellaneous viruses—canine distemper virus, parvovirus

Uterine

- Cystic endometrial hyperplasia (CEH, a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts) and inflammation with accumulation of pus in the uterus (known as “pyometra”)
- Trauma
- Tumors or cancer
- Medications that are toxic to the developing embryo
- Chemotherapeutic agents

- Estrogens
- Steroids—high dosages

Ovarian

- Prostaglandins—substances that have many effects on the female reproductive tract, one of which is the breakdown of the “corpora luteum” or “yellow body” that develops at the site of ovulation in the ovary and produces the female hormone, progesterone, which supports and maintains the pregnancy; breakdown or lysis of the corpora luteum decreases levels of progesterone and disrupts support of pregnancy
- Dopamine agonists—medications that mimic dopamine (a nervous system “messenger”) that leads to a decrease in the hormone, prolactin, and to lysis of the “corpora luteum” or “yellow body” via suppression of prolactin; drugs include bromocriptine and cabergoline
- Insufficient secretion of progesterone by the “corpora luteum” or “yellow body” during pregnancy, leading to pregnancy loss (known as “hypoluteoidism”)—abnormal function of the corpora luteum in the absence of fetal, uterine, or placental disease: progesterone concentrations less than 1 to 2 ng/ml

Hormonal Dysfunction

- Low levels of thyroid hormone (hypothyroidism)
- Excessive levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Environmental factors—hormone or endocrine-disrupting contaminants have been documented in people and wildlife with fetal loss

Fetal Defects

- Lethal genetic or chromosomal abnormality
- Lethal organ defects

RISK FACTORS

- Exposure of the brood bitch to animals carrying disease
- Old age
- Genetic factors



TREATMENT

HEALTH CARE

- Most bitches should be confined and isolated pending diagnosis
- Hospitalization of infectious patients preferred
- *Brucella canis*—highly infective to dogs; bacteria shed in high numbers during abortion; suspected cases should be isolated
- Outpatient medical management—medically stable patients with noninfectious causes of pregnancy loss, hormonal disorders, or disease of the lining of the uterus (known as “endometrial disease”)
- Partial abortion—may attempt to salvage any remaining live fetuses; administer antibiotics if a bacterial component is identified
- Dehydration—use replacement fluids, supplemented with electrolytes if imbalances are identified by serum biochemistry blood tests

ACTIVITY

- Partial abortion—cage rest

DIET

- No special dietary considerations for uncomplicated cases

SURGERY

- Spay or ovariectomy—preferred for stable patients with no breeding value



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Prostaglandin (PGF_{2α}—Lutalyse®)—stimulates and evacuates the uterus; also may consider Estrumate® (cloprostenol)—not approved for use in dogs; discuss the risks and benefits of treatment with your dog’s veterinarian
- Antibiotics—for bacterial disease; initially institute broad-spectrum antibiotic; specific antibiotic depends on bacterial culture and sensitivity testing of vaginal tissue or postmortem examination of fetus(es)
- Progesterone (Regu-Mate®) or progesterone in oil—for documented cases of insufficient secretion of progesterone (female hormone necessary to support pregnancy) by the “corpora luteum” or “yellow body” (hypoluteoidism) only
- Oxytocin (hormone that stimulates uterine contractions)—for uterine evacuation; most effective in the first 24 to 48 hours after abortion



FOLLOW-UP CARE

PATIENT MONITORING

- Partial abortion—monitor remaining fetuses with ultrasound examination to determine if they are continuing to live and develop; monitor general (systemic) health of the bitch for remainder of pregnancy
- Vulvar discharges—check daily; for decreasing amount, odor, and inflammatory component; for consistency (increasing mucoid content is good prognostically)
- Prostaglandin (PGF_{2α})—continued for 5 days or until most of the discharge ceases (usually 3 to 15 days)
- *Brucella canis*—monitor after spaying and antibiotic therapy; yearly serum testing to identify reappearance of bacteria (extremely difficult to eliminate infection successfully, even if combined with spaying or ovariectomy)
- Low levels of thyroid hormone (hypothyroidism)—treat appropriately; spaying recommended (possible genetic nature of hypothyroidism should be considered)

PREVENTIONS AND AVOIDANCE

- Brucellosis (disease caused by *Brucella canis*) and other infectious agents—surveillance programs to prevent introduction to kennel
- Spay or ovariectomy—for bitches with no breeding value

POSSIBLE COMPLICATIONS

- Untreated inflammation with accumulation of pus in the uterus (pyometra)—generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”), presence of poisons or toxins in the blood (known as “toxemia”), death
- Brucellosis (disease caused by *Brucella canis*)—infection and inflammation in other organs of the body, such as the vertebrae (diskospondylitis) and eye (endophthalmitis, recurrent uveitis)

EXPECTED COURSE AND PROGNOSIS

- Inflammation with accumulation of pus in the uterus (pyometra)—recurrence rate during subsequent cycle is high (up to 70%) unless pregnancy is established
- Cystic endometrial hyperplasia (CEH, a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts)—recovery of fertility unlikely; and inflammation with accumulation of pus in the uterus (pyometra) is a common complication
- Hormonal dysfunction—often manageable; familial (runs in certain families or lines of animals) aspects should be considered
- Brucellosis (disease caused by *Brucella canis*)—guarded prognosis; extremely difficult to eliminate infection successfully, even if combined with spaying or ovariectomy



KEY POINTS

- If brucellosis (disease caused by *Brucella canis*) is confirmed as the cause of pregnancy loss, euthanasia is recommended owing to lack of successful treatment and to prevent spread of infection; may try spay or ovariectomy and long-term antibiotics with long-term monitoring
- If brucellosis (disease caused by *Brucella canis*) is confirmed as the cause of pregnancy loss, a surveillance program for kennel situations should be developed and implemented
- If brucellosis (disease caused by *Brucella canis*) is confirmed as the cause of pregnancy loss, zoonotic potential should be considered; a “zoonosis” is a disease that can be passed from animals to people
- Primary uterine disease—spay or ovariectomy is indicated in patients with no breeding value; cystic endometrial hyperplasia (CEH, a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts) is an irreversible change
- Infertility or pregnancy loss—may recur in subsequent “heat” or “estrous” cycles despite successful immediate treatment
- Prostaglandin treatment—discuss possible side effects of prostaglandins with your pet’s veterinarian
- Infectious diseases—establish surveillance and control measures

ABORTION, TERMINATION OF PREGNANCY in DOGS



BASICS

OVERVIEW

- Intentional medical or surgical termination of an unwanted pregnancy in a dog; may be accomplished by using drugs that prevent fertilization of the egg or prevent implantation of the embryo in the uterus or by using drugs or surgery to terminate an established pregnancy

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Unwanted pregnancy in any breed

Predominant Sex

- Female

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on the stage of the pregnancy (gestation)
- May have no visible signs of pregnancy
- Discharge of fluid or developing fetus(es) from female genital canal (vagina)

CAUSES

- Medically stopping development of the “corpus luteum” or “yellow body” in the ovary that produces the female hormone progesterone, which supports and maintains the pregnancy
- Inhibiting the production of progesterone, the female hormone that supports pregnancy
- Using a drug to block the effects of progesterone, the female hormone that supports pregnancy

RISK FACTORS

- Drugs used to terminate a pregnancy may have undesirable side effects
- Medical treatment may require a great deal of time and effort
- Anesthesia and surgical risks if ovariohysterectomy (surgical removal of the ovaries and uterus, also known as a “spay”) is performed



TREATMENT

HEALTH CARE

- Inpatient care is preferred to allow for monitoring of the patient; the patient should be monitored for at least 1 hour prior to discharge if the owner wishes to take the patient home
- Many accidentally mated dogs do not become pregnant; therefore, treatment may not be necessary
- Determining pregnancy status in the early stages is difficult because ultrasound confirmation of pregnancy is not possible until 4–5 weeks after breeding
- Medical treatment is generally done early in the pregnancy as later treatment may lead to more discharge and possible visually detected passage of fetuses

ACTIVITY

- No need to change the patient’s activity following medical treatment
- Activity and exercise is restricted for several days following surgery if ovariohysterectomy (spay) is performed

DIET

- Delay feedings for at least 1–2 hours after medical treatments—reduces nausea and vomiting
- Follow feeding instructions before and after surgery if ovariohysterectomy (spay) is performed

SURGERY

- If breeding is not a consideration, ovariohysterectomy (spay) may be the best alternative treatment



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Prostaglandin $F_{2\alpha}$ or $PGF_{2\alpha}$ (Lutalyse)—causes breakdown of the corpus luteum and causes cervical dilation and uterine contractions
- Bromocriptine mesylate (Parlodel)—causes abortion and inhibits hormone, prolactin, that stimulates milk production
- Cabergoline (Dostinex)—inhibits hormone, prolactin, that stimulates milk production
- Prostaglandin $F_{2\alpha}$ or $PGF_{2\alpha}$ combined with bromocriptine or cabergoline
- Dexamethasone—mode of action not known
- Mifepristone (blocks the effects of progesterone) and epostane (prevents production of progesterone) are potentially useful; not currently available to veterinarians in North America



FOLLOW-UP CARE

PATIENT MONITORING

- Examination of uterus using ultrasound to confirm emptying of uterine contents

PREVENTIONS AND AVOIDANCE

- Surgical sterilization (ovariohysterectomy or spay) for dogs not intended for breeding
- Confine dogs intended for breeding during heat cycle, walk on leash, and observe carefully to avoid accidental breeding

POSSIBLE COMPLICATIONS

- Medical treatment may shorten time until next heat cycle
- Estrogen compounds should not be used as treatment to cause abortion
- Bleeding, infection, and incision problems may occur following ovariohysterectomy or spay

EXPECTED COURSE AND PROGNOSIS

- Fertility may be preserved with medical treatment
- Ovariohysterectomy (spay) will eliminate heat cycles and fertility



KEY POINTS

- If breeding is not a consideration, ovariohysterectomy (spay) may be the best alternative
- Discuss all treatment options with the veterinarian, and come to a mutually agreeable treatment plan

ABSCCESS



BASICS

OVERVIEW

- An abscess is a localized collection of pus contained within a cavity somewhere in the body

SIGNALMENT/DESCRIPTION of ANIMAL

- Cats and dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Determined by organ system and/or tissue affected
- A rapidly appearing, painful swelling with or without discharge (if affected area is visible)
- Associated with a combination of inflammation (seen as pain, swelling, redness, heat, and loss of function), tissue destruction, and/or organ system dysfunction caused by accumulation of pus
- A discrete mass of varying size may be detectable; the mass may be firm or fluid-filled
- Inflammation and discharge from a draining tract may be visible if the abscess is superficial and has ruptured to an external surface
- Fever, if abscess is not ruptured and draining
- Generalized bacterial infection (sepsis) occasionally, especially if abscess ruptures internally

CAUSES

- Trauma (such as fight wounds) or previous infection
- Foreign objects
- Pus-causing (pyogenic) bacteria—*Staphylococcus*; *Escherichia coli*; β -hemolytic *Streptococcus*; *Pseudomonas*; *Mycoplasma* and *Mycoplasma*-like organisms (L-forms); *Pasteurella multocida*; *Corynebacterium*; *Actinomyces*; *Nocardia*; *Bartonella*
- Bacteria that can only live and grow in the absence of oxygen (known as “obligate anaerobic bacteria”)—*Bacteroides*; *Clostridium*; *Peptostreptococcus*; *Fusobacterium*

RISK FACTORS

Risk factors for the formation of an abscess are determined by the organ system and/or tissue affected. The following organs and tissues are listed with their risk factors:

- Anal sac—impaction; anal sac inflammation
- Brain—inner ear infection (otitis interna); sinus infection (sinusitis); infection in the mouth (oral infection)
- Liver—inflammation of the umbilical veins (omphalophlebitis); generalized bacterial infection (sepsis)
- Lung—foreign object aspiration; bacterial pneumonia
- Mammary gland—mastitis
- Tissues around the eye (periorbital tissues)—dental disease; chewing of wood or other plant material
- Skin—fighting (fight wounds)
- Prostate gland—bacterial infection of the prostate (bacterial prostatitis)
- Immunosuppression (diseases or drug therapy that lead to an inability to develop a normal immune response)—feline leukemia virus (FeLV) or feline immunodeficiency virus (FIV) infection, immunosuppressive chemotherapy, acquired or inherited immune system dysfunctions, underlying predisposing disease (such as diabetes mellitus, chronic kidney failure, hyperadrenocorticism [condition in which the adrenal glands produce excessive steroids])



TREATMENT

HEALTH CARE

- Depends on location of abscess and treatment required
- Outpatient—bite-induced abscesses
- Inpatient—generalized bacterial infection (sepsis); extensive surgical procedures; treatment requiring extended hospitalization
- Establish and maintain adequate drainage of pus
- Surgical removal of the center of the infection (known as the “nidus”) or foreign object(s), if present
- Appropriate antimicrobial or antibiotic therapy; length of time for antibiotic therapy varies based on the bacteria causing the infection and the location of the abscess/infection
- Apply warm compresses or packs to inflamed area as directed by your pet’s veterinarian
- Use protective bandaging and/or Elizabethan collars as directed by your pet’s veterinarian
- Accumulated pus—the veterinarian will drain the abscess and maintain drainage by medical and/or surgical means
- Generalized bacterial infection (sepsis) or bacterial infection of the lining of the abdomen (peritonitis)—aggressive fluid therapy and support

ACTIVITY

- Restrict until the abscess has resolved and adequate healing of tissues has taken place

DIET

- Sufficient nutritional intake is required to promote healing and recovery
- Depends on location of abscess and treatment required

SURGERY

- Appropriate removal of infected tissue (débridement) and drainage of the abscess—may need to leave the wound open to an external surface of the body to promote drainage; may need to place surgical drains
- Early drainage—to prevent further tissue damage and formation of abscess wall
- Remove any foreign object(s), dead (necrotic) tissue, or center (nidus) of infection



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antimicrobial drugs or antibiotics—effective against the infection-causing bacteria; gain access to site of infection
- Broad-spectrum antimicrobial drugs or antibiotics—kill many types of bacteria (bactericidal) with activity against bacteria that can live and grow in the presence of oxygen (aerobic bacteria) and bacteria that can live and grow in the absence of oxygen (anaerobic bacteria) until results of bacterial culture and sensitivity are known; dogs and cats: amoxicillin; amoxicillin/clavulanic acid; clindamycin; and trimethoprim/sulfadiazine; cats with *Mycoplasma* and L-forms: doxycycline
- Aggressive antimicrobial therapy is required for generalized bacterial infection (sepsis) or bacterial infection of the lining of the abdomen (peritonitis)



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor for progressive decrease in drainage, resolution of inflammation, and improvement of clinical signs

PREVENTIONS AND AVOIDANCE

- Anal sac abscesses—prevent impaction; consider surgical removal of the anal sacs (anal saculectomy) for repeated episodes of anal sac abscess
- Mastitis—prevent lactation (such as by spaying)
- Abscesses in the tissues around the eye—do not allow chewing on foreign object(s)
- Skin abscesses—prevent fighting
- Prostatic abscesses—castration possibly helpful

POSSIBLE COMPLICATIONS

- Generalized bacterial infection (sepsis)
- Inflammation of the lining of the abdomen (peritonitis) and/or inflammation of the lining of the chest (pleuritis) if an abscess located in the abdomen or chest ruptures
- Compromise of organ function
- Delayed drainage and treatment may lead to chronically draining tracts

EXPECTED COURSE AND PROGNOSIS

- Depend on organ system involved and amount of tissue destruction



KEY POINTS

- Correct or prevent risk factors
- Adequate drainage of the abscess
- Surgical removal of the center of the infection (known as the “nidus”) or foreign object(s), if present
- Start appropriate antimicrobial or antibiotic therapy; length of time for antibiotic therapy varies based on the bacteria causing the infection and the location of the abscess/infection

ACETAMINOPHEN TOXICITY



BASICS

OVERVIEW

- Results from owners overdosing the patient with over-the-counter medications containing acetaminophen, a medication intended to control pain or fever in humans

GENETICS

- Cats—genetic deficiency in a pathway that breaks down or changes (metabolizes) drugs in the liver (known as the “glucuronide conjugation pathway”); makes cats vulnerable to acetaminophen toxicity

SIGNALMENT/DESCRIPTION of ANIMAL

- Cats; dogs
- Most common drug toxicity in cats; considerably less frequent in dogs
- Young and small dogs and cats—greater risk from owner-given single-dose acetaminophen medications

SIGNS/OBSERVED CHANGES in the ANIMAL

- May develop 1–4 hours after dosing
- Progressive depression
- Rapid breathing
- Darkened mucous membranes (moist tissues of body, such as gums)
- Drooling (salivation)
- Vomiting
- Abdominal pain
- Rapid breathing (tachypnea) and bluish discoloration of skin and moist tissues of body (cyanosis) due to a abnormal compound (methemoglobin) in the blood (methemoglobinemia) that disrupts the ability of the red blood cells to carry oxygen to the body
- Fluid build up (edema)—face, paws, and possibly forelimbs; after several hours
- Chocolate-colored urine due to the presence of blood in the urine (hematuria) and the presence of methemoglobin in the urine (methemoglobinuria); especially in cats
- Death

CAUSES

- Acetaminophen overdosing

RISK FACTORS

- Nutritional deficiencies of glucose and/or sulfate
- Simultaneous administration of other glutathione-depressing drugs



TREATMENT

HEALTH CARE

- With methemoglobinemia—must evaluate promptly; inpatient care
- With dark or bloody urine or yellowish discoloration of skin and moist tissues of the body (jaundice or icterus)—inpatient care
- Gentle handling—imperative for clinically affected patients
- The veterinarian will induce vomiting (emesis) and may perform flushing of the stomach (gastric lavage)—useful within 4–6 hours of ingestion of acetaminophen
- Low blood count (anemia), blood in the urine (hematuria), or presence of hemoglobin in the urine (hemoglobinuria)—may require whole blood transfusion
- Fluid therapy to maintain hydration and electrolyte balance
- Drinking water should be available at all times

ACTIVITY

- Restricted

DIET

- Food—offered 24 hours after initiation of treatment



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Activated charcoal—administered immediately after the veterinarian has induced vomiting or flushed the stomach (gastric lavage) and after vomiting is controlled; activated charcoal is used to attract and keep the remaining acetaminophen in the gastrointestinal tract
- *N*-acetylcysteine (Mucomyst) is administered; considered to be an antidote for acetaminophen toxicity
- Other sulfur donor drugs—if *N*-acetylcysteine not available; sodium sulfate
- 1% methylene blue solution—combats methemoglobinemia without inducing red blood cell destruction (known as a “hemolytic crisis”)
- Ascorbic acid—slowly reduces methemoglobinemia



FOLLOW-UP CARE

PATIENT MONITORING

- Continual clinical monitoring of methemoglobinemia
- Serum liver enzyme activities to monitor liver damage
- Blood glutathione level—provide evidence of the effectiveness of therapy

PREVENTIONS AND AVOIDANCE

- Never give acetaminophen to cats
- Give careful attention to acetaminophen dose in dogs; acetaminophen should only be given to dogs under a veterinarian’s supervision

POSSIBLE COMPLICATIONS

- Liver damage and resulting scarring (fibrosis)—may compromise long-term liver function in recovered patients

EXPECTED COURSE AND PROGNOSIS

- Rapidly progressive methemoglobinemia—serious sign
- Methemoglobin concentrations greater than 50%—grave prognosis
- Progressively rising serum liver enzymes 12–24 hours after ingestion—serious concern
- Expect clinical signs to persist 12–48 hours; death owing to methemoglobinemia possible at any time
- Dogs and cats receiving prompt treatment that reverses methemoglobinemia and prevents excessive liver damage may recover fully
- Dogs—death as a result of liver damage may occur in a few days
- Cats—death as a result of methemoglobinemia occurs 18–36 hours after ingestion



KEY POINTS

- Never give acetaminophen to cats
- Acetaminophen should only be given to dogs under a veterinarian’s supervision
- Most common drug toxicity in cats; considerably less frequent in dogs
- Treatment in clinically affected patients may be prolonged and expensive
- Patients with liver injury may require prolonged and costly management

AGGRESSION BY DOGS TOWARD FAMILIAR PEOPLE



BASICS

OVERVIEW

- Aggression (such as growling, lip-lifting, barking, snapping, lunging, biting), usually directed toward household members or familiar people in situations involving access to preferred resources (such as food or toys)
- Also referred to as “dominance aggression,” “status-related aggression,” “conflict aggression,” or “competitive aggression”

GENETICS

- Breed bias or predilections exist and pedigree analyses have shown that it may occur more commonly within related dogs
- Mode of inheritance is unknown

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog

Breed Bias or Predilections

- Spaniels (English springer and cocker), terriers, Lhasa apso, and rottweiler, but may be exhibited by any breed

Mean Age And Range

- Usually manifested at the onset of social maturity (12–36 months of age); may be seen in young dogs

Predominant Sex

- Male dogs (castrated and intact) more commonly are presented with aggression toward familiar people than are female dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Aggression often seen around resting areas, food, toys, handling (including petting) and reaching toward favorite possessions, including people
- Aggression usually is directed toward household members or persons that have an established relationship with the dog
- Aggressive behaviors may be seen in other contexts, including (but not limited to) defense of territory, when dogs are reprimanded or denied access to items or activities, toward other dogs, and toward unfamiliar people
- Aggression may not be seen every time dog is in a certain situation and may not be directed uniformly toward each person within the household
- Stiff body posture, staring, head up, ears up and forward, or tail up usually accompanies aggressive behavior; a combination of these postures may be seen with more submissive postures (for example, tail is up but ears are tucked, eyes averted), which may represent an element of conflict, anxiety, or fear in the dog’s motivation
- Dog may seem to be “moody” and this behavior may be a key to judging when the dog is likely to be aggressive in a given situation
- Dog may show fearful behaviors (such as eye aversion, tail tucked, and avoidance) in early episodes; these fearful behaviors may diminish as the dog becomes more confident that aggression will change the outcome of the situation
- Anxiety may be noted in pet-owner interactions and other situations, such as owner departure or novel situations
- Some dogs control their environment using aggression only because it is effective, but are anxious about every encounter, while other dogs appear confident and secure
- Generally the dog does not have any physical abnormalities related to the aggressive behavior; however, medical conditions, especially painful ones, may contribute to the expression of aggression

CAUSES

- May be part of a normal canine social behavioral repertoire, but its expression is influenced by environment, learning, and genetics
- The manifestation of aggression may be influenced by underlying medical conditions, early experiences (learning that aggression works to control situations), inconsistent or lack of clear rules and routine within the household and within human-pet interactions
- Rarely a sign of a medical condition, but contributory medical conditions must be ruled out, since illness and/or pain may influence a tendency for aggressive behaviors

RISK FACTORS

- Inconsistent or inappropriate punishment and inconsistent owner interactions may contribute to the development of conflicted and/or aggressive behavior
- Medical conditions, especially painful ones, may contribute to the expression of aggression; extreme caution should be taken when the veterinarian examines dogs that show aggression—including the use of muzzles or other humane restraint devices



TREATMENT

HEALTH CARE

- Outpatient behavior modification and possibly medical management
- Avoid situations that might evoke aggression; identify specific situations to avoid—do not allow the dog on furniture; do not give valuable treats or toys (such as rawhides); pick up toys and control playtime and activity; limit physical contact with the dog, including petting
- Do not physically punish or reprimand the dog
- Teach the dog to comfortably and safely wear a head halter (such as a Gentle Leader®) and/or basket muzzle; have the dog wear the head halter with a lightweight 8–10 foot leash attached whenever in contact with people
- Use a long leash to move the dog from situations that may elicit aggression; do not reach for dog directly
- Behavior modification—use non-confrontational methods to teach the dog to view people as leaders; use reward-based training techniques to teach the dog to obey commands from people without the dog experiencing conflict or becoming aggressive
- Affection control—make the dog follow a command before getting anything it desires from people (also known as “Nothing in life is free” or “Learn to earn”)—for example, the dog must sit or lie down before feeding, petting, play, or going for a walk
- For initial 2–3 week period, owners should give the dog attention only during brief, structured (for example, command-response-reward) periods; at other times, they must ignore the dog, especially if it is soliciting attention
- Counter commanding—using positive reinforcement (such as food, toys, play, petting) to teach behaviors that are counter to those that have resulted in aggression in the past—for example, teach an “off” command to move off furniture or “drop it” command to release toys
- Desensitization and counterconditioning—technique used to decrease responsiveness to situations that have resulted in aggression in the past (dog may need to be muzzled for safety); should not begin until the owner has assumed a greater level of control over the dog through affection control and reward-based training

ACTIVITY

- Appropriate physical activity may help decrease incidences of aggression

DIET

- Low protein/high tryptophan diets may help reduce aggression, but are unlikely to make a significant difference without behavior modification

SURGERY

- Neuter intact males
- Females that start to show dominance aggression at less than 6 months of age may be less aggressive when mature if not spayed



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No medications are approved by the FDA for the treatment of canine aggression
- Owners must be aware that the use of a medication is off-label and be informed of potential risks and potential side effects
- Signed informed consent forms are prudent
- Before prescribing medication be sure that owners understand the risks involved in owning an aggressive dog and will follow safety procedures and not rely on medication to keep others safe
- Never use medications without behavior modification
- Medication may not be appropriate in some family situations, such as those with small children, family members with disabilities, or immunocompromised individuals, who are unable to develop a normal immune response
- Drugs that have been used in treating canine aggression include Selective Serotonin Reuptake Inhibitors (SSRIs), such as fluoxetine, paroxetine, sertraline; Tricyclic Antidepressants (TCAs), such as clomipramine; and progestins, such as DepoProvera



FOLLOW-UP CARE

PATIENT MONITORING

- Owners often need ongoing assistance with behavior cases, especially aggression
- At least one follow-up call within the first 1–3 weeks after the consultation is advisable; provisions for further follow-up either by

phone or in person should be made at that time

PREVENTIONS AND AVOIDANCE

- Continued avoidance of situations that lead to aggression (known as “aggression triggers”) may be necessary

POSSIBLE COMPLICATIONS

- Human injuries; surrender of dog to animal control or animal shelter; euthanasia of dog

EXPECTED COURSE AND PROGNOSIS

- No cure exists; prognosis for improvement is better if aggression is at a low intensity and in relatively few predictable situations; prognosis is highly dependent on owner compliance
- Treatment recommendations are lifelong
- Owners may see recurrence of aggression with treatment lapses



KEY POINTS

- Successful treatment, as measured by a decrease in aggressive incidents, depends upon the owner’s understanding of basic canine social behavior, the risks involved in living with an aggressive dog, and how to implement safety and management recommendations
- Preventing human injuries must be the first concern
- Treatment is aimed at controlling the problem, not at achieving a “cure”
- Owners must be aware that the only way to prevent future injuries is euthanasia
- It is very important that owners are educated about the risks of using physical punishment and training techniques that rely upon owners learning to “dominate” their dogs; the improper use of physical punishments/dominance techniques ranging from corrections with choke chains to so-called “alpha rolls” to setting up situations to provoke and then correct aggression can lead to human injury, increased aggression, an increase in underlying anxiety and a disruption of the human-animal bond

AGGRESSION IN CATS: OVERVIEW



BASICS

OVERVIEW

- Aggression can be an appropriate behavior that allows the cat to protect itself (known as an “adaptive behavior”) and its resources (such as food)
- Behavioral medicine—concerned with recognizing when aggressive behavior is abnormal or inappropriate (known as “maladaptive behavior”)
- Numerous types of aggression have been identified in cats, including the following:
 1. Aggression owing to lack of socialization
 - No human contact before 3 months of age—cat misses sensitive period important for development of normal approach responses to people; if not handled until 14 weeks of age, it usually is fearful and aggressive to people; if handled for only 5 min/day until 7 weeks of age, it interacts with people, approaches inanimate objects, and plays with toys.
 - Lack of social interaction with other cats—may result in lack of normal inquisitive response to other cats
 - These cats are usually not normal, cuddly pets; they may eventually attach to one person or a small group of people; if forced into a situation involving restraint, confinement, or intimate contact, they may become extremely aggressive
 2. Play aggression
 - Weaned early and hand-raised by humans—cat may never learn to temper its play responses; if not taught as a kitten to modulate responses, it may not learn to sheathe claws or inhibit bite; bottle-fed cats may be over represented
 3. Fearful or fear-induced aggression
 - Fearful—cat may hiss, spit, arch the back, and hair may stand up if flight is not possible; combinations of offensive and defensive postures and overt and covert aggressive behaviors are usually involved
 - Flight—virtually always a component of fearful aggression if the cat can escape
 - Pursued—if cornered, cat will stop, draw its head in, crouch, growl, roll on its back when approached (not submissive but overtly defensive), and paw at the approacher; if pursuit is continued, cat will strike, then hold the approacher with its forepaws while kicking with the back feet and biting
 - If threatened, cat will defend itself; any cat can become fearfully aggressive
 4. Pain aggression
 - Pain may cause aggression; with extended painful treatment, cat may exhibit fearful aggression
 5. Cat-to-cat (intercat) aggression
 - Male cat-to-male cat aggression associated with mating or hierarchical status within the social group; mating also may involve social hierarchy issues
 - Maturity—in peaceful multi-cat households, problems may occur, regardless of sexes in the household, when a cat reaches social maturity (2–4 years of age)
 6. Maternal aggression
 - May occur during the period surrounding the birth of kittens (known as the “periparturient period”)
 - Protection—queens may guard nesting areas and kittens by threatening with long approach distances, rather than attack; usually directed toward unfamiliar individuals; may inappropriately be directed toward known individuals; as kittens mature, aggression resolves
 - Unknown if kittens learn aggressive behavior from an aggressive mother
 7. Predatory behavior
 - Occurs under different behavioral circumstances
 - Normal predatory behavior develops at 5–7 weeks of age; kitten may be a proficient hunter by 14 weeks of age; commonly displayed with field voles, house mice, and birds at feeders; may be learned from mother; more common in cats that have to fend for themselves; if well fed, cat may kill prey without feeding on it
 - Aggression—stealth, silence, heightened attentiveness, body posture associated with hunting (slinking, head lowering, tail twitching, and pounce postures), lunging or springing at prey, exhibiting sudden movement after a quiet period
 - In free-ranging groups of cats, when a new male enters, he may kill kittens to encourage queen to come into heat (estrus)
 - Inappropriate context distinctions about prey—potentially dangerous if “prey” is a foot, hand, or infant; cats exhibiting pre-pounce behaviors in these contexts are at risk of displaying inappropriate predatory behavior
 8. Territorial aggression
 - May be exhibited toward other cats, dogs, or people; owing to transitive nature of social hierarchies, a cat aggressive to one housemate may not be to another if its turf is not contested
 - Turf may be delineated by patrol, chin rubbing, spraying, or non-spraying marking; threats and/or fights may occur if a perceived offender enters the area; if the struggle involves social hierarchy, the challenger may be sought out and attacked after the territory is invaded
 - May be difficult to treat, particularly if the cat is marking its territory; marking problems suggest a possible

- underlying aggression
9. Redirected aggression
 - Difficult to recognize and may be reported as incidental to another form of aggression
 - Occurs when a aggressive behavior pattern appropriate for a specific motivational state is redirected to an accessible target because the primary target is unavailable (e.g., cat sees a bird outside the window and is demonstrating predatory behavior; person walks behind cat and cat pounces the person, possibly biting the person); cat may remain reactive for some time after being thwarted in an aggressive interaction
 - Often precipitated by another inappropriate behavior or event; important to treat that behavior as well
 10. Assertion or status-related aggression
 - If unprovoked, most frequently occurs when cat is being petted; a need to control all interactions with humans and when attention starts and ceases; cat may bite and leave or may take hand in teeth but not bite
 - May be accompanied by true territorial aggression where specific areas are patrolled
 - May best be called impulse control/dyscontrol aggression
 - Exact syndrome is not well defined or recognized
 11. Idiopathic aggression
 - Rare; poorly understood and poorly defined; unprovoked, unpredictable, “toggle-switch” (turned on and off) aggression

SIGNALMENT/DESCRIPTION of ANIMAL

- Any breed of cat
- Some types of aggression appear at onset of social maturity (2–4 years)
- Males—may be more prone to cat-to-cat aggression (known as “intercat” aggression)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Aggressive behavior
- Physical examination findings are generally secondary to aggression, such as injuries, lacerations, or damage to teeth or claws
- Continuous anxiety—decreased or increased grooming

CAUSES

- Aggression is part of normal feline behavior; greatly influenced by the early social history and exposure to humans and other animals, sex, social context, handling, and many other variables

RISK FACTORS

- Abuse—cat may learn aggression as a pre-emptive strategy to protect itself



TREATMENT

HEALTH CARE

- Desensitization, counterconditioning, flooding, and habituation—if subtleties of social systems and communication are understood



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

No drugs are approved by the FDA for the treatment of aggression in cats; your veterinarian will discuss the risks and benefits of medical treatment

- Antianxiety medications that increase levels of serotonin in the central nervous system, such as tricyclic antidepressants (TCAs) and selective serotonin reuptake inhibitors (SSRIs)
- Amitriptyline (TCA)
- Imipramine
- Buspirone; may make some cats more assertive; thus may work well for the victim in anxiety-associated aggression
- Clomipramine (TCA)
- Fluoxetine or paroxetine (SSRI)
- Buspirone, clomipramine, paroxetine and fluoxetine—may take 3–5 weeks to be fully effective; early effects in cats are seen within 1 week, best for active, overt aggressions
- Nortriptyline—active intermediate metabolite of amitriptyline
- Anxious and fearful aggression combined with elimination disorders (behavioral problems involving urination and/or defecation)—diazepam or other benzodiazepine; use with caution because benzodiazepines can worsen inhibited aggressions; may facilitate some behavior modification if food treats used



FOLLOW-UP CARE

PATIENT MONITORING

- Blood work (complete blood count, serum biochemistry) and urinalysis should be performed before treatment; semiannually in older patients; yearly in younger patients if treatment is continuous; adjust dosages accordingly
- As warranted by clinical signs—vomiting; gastrointestinal distress; rapid heart rate (tachycardia), and rapid breathing (tachypnea)

PREVENTIONS AND AVOIDANCE

- Ensure appropriate socialization of kittens with humans and other cats
- Avoid provocation of the cat
- Observe signs of aggression (such as tail flicking, ears flat, pupils dilated, head hunched, claws possibly unsheathed, stillness or tenseness, low growl) and safely interrupt the behavior; leave cat alone and refuse to interact until appropriate behavior is displayed; if the cat is in the person's lap, let the cat drop from his or her lap
- Discourage direct physical correction; may intensify aggression
- If possible, safely separate cats; keep the active aggressor in a less favored area to passively reinforce more desirable behavior
- Remember that a cat displaying aggressive or predatory behavior can bite or scratch any person or another animal--always be careful to ensure that you do not get injured; the best approach in some situations is to leave the cat alone in a quiet area until it calms down

POSSIBLE COMPLICATIONS

- Human injuries; surrender of cat to animal control or animal shelter; euthanasia of cat
- Left untreated, these disorders always progress



KEY POINTS

- Aggression can be an appropriate behavior that allows the cat to protect itself (known as an “adaptive behavior”) and its resources (such as food)
- Behavioral medicine is concerned with recognizing and identifying abnormal or inappropriate aggressive behavior
- Numerous types of aggression have been identified in cats
- Early treatment using both behavioral modification and pharmacological intervention is crucial
- Left untreated, these disorders always progress

OVERVIEW OF AGGRESSION IN DOGS



BASICS

OVERVIEW

- Action taken by one dog directed against a person or another animal, with the result of harming, limiting, or depriving that person or animal; aggression may be offensive, defensive, or predatory (that is, hunting behavior)
- Offensive aggression—unprovoked attempt to gain some resource (such as food or toys) at the expense of another; includes social status/dominance, inter-male (that is, between two males), and inter-female (that is, between two females) aggression
- Defensive aggression—aggression by a “victim dog” toward a person or another animal that is perceived as an instigator or threat; includes fear-motivated, territorial, protective, irritable (pain-associated or frustration-related), and maternal aggression
- Predatory aggression—rare; the dog’s aggression may be triggered by “prey” behavior by the victim (person or another animal), such as running or squealing

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Any breed may show aggression
- Pit bulls, rottweilers, German shepherd dogs—associated with fatal dog bites

Mean Age and Range

- Any age puppy or dog may show aggression
- Social status/dominance-related offensive aggression—escalates near the time the dog reaches social maturity (1 to 2 years of age)

Predominant Sex

- Any sex dog may show aggression
- Males—intact or castrated
- Females—intact; maternal aggression

SIGNS/OBSERVED CHANGES in the ANIMAL

- Behavioral warning signs include being motionless (immobility), growling, snarling, or snapping at air; offensive aggression warning signs include head up, tail up, direct stare, face-on immobility; defensive aggression warning signs include head lowered, tail down, and body withdrawn
- Physical examination usually unremarkable
- Dominance-related aggression, fear-related aggression, or irritable aggression may be evident during the examination
- Nervous system examination—abnormalities may suggest a disease process (such as rabies) as the cause of aggression
- Signs vary, according to the situation and the type of aggression

Social Status/Dominance Aggression

- Directed toward household members
- Head up; tail up; staring; stiff gait
- Triggers that stimulate aggression include reaching for pet, patting on head, pushing off sleeping sites, approaching food or stolen objects
- Also called “conflict aggression”

Inter-male (between two males) and Inter-female (between two females) Aggression

- Directed toward other dogs, usually same sex
- Human injury, when person interferes with fights
- Head up; tail up; staring; stiff gait

Fear-Motivated Aggression

- Directed to people or dogs who approach, stand over, or reach for the dog
- Certain familiar people may be exempt
- No gender bias
- Head down; eyes wide; tail tucked, spine in “C” curve

Territorial Aggression

- Directed toward strangers approaching home, yard, or car
- May be increased in intensity, if the dog is restrained
- Agitation, barking; lunging; baring teeth
- Approach/avoidance behavior is common; “approach/avoidance” behavior consists of the dog approaching the stranger and then moving back away from the stranger

Protective Aggression

- Directed toward stranger approaching owner
- Escalates with decreasing distance between the stranger and the dog and owner

Irritable (Pain, Frustration) Aggression

- Restricted to specific context associated with pain (for example, nail trim, injection) or conflict associated with being restrained
- Other forms of aggression (social status/dominance and fear-induced aggression) should be considered as possible causes of signs

Maternal Aggression

- Directed toward individuals approaching the whelping area or puppies
- Intensity usually related to age of puppies; the intensity of aggression is greater the younger the puppies

CAUSES

- Part of the normal range of dog behavior; strongly influenced by breed, sex, early socialization, handling, and individual temperament
- May be caused by a medical condition—possible but rare; medical causes of aggression should be considered in all cases

RISK FACTORS

- Poor socialization to certain types of stimuli (such as children)—adult dog may display fear-related aggression
- Environmental conditions may lead to aggression or may increase the level of aggression—such as associating with other dogs in a pack; barrier frustration or tethering; cruel handling and abuse; and dog baiting and fighting



TREATMENT

HEALTH CARE

- The first tenet of management is to prevent injury to people
- Overtly aggressive dogs are never cured, occasionally they may be managed successfully
- Euthanasia—appropriate solution in cases of vicious dogs; may be the only safe solution
- Board the dog until an outcome decision or implementation of a safe management plan is made
- Use physical barriers, to reduce risk of injury to people, until the owner obtains treatment
- Identify specific situations that have led to aggression in the past; use a specific plan to avoid these situations
- Improve physical control of the dog using reliable barriers (such as fences, baby gates), muzzles, leashes, and head halters
- Calmly and safely remove dog from aggressive-provoking situations
- Avoid punishment and confrontation; punishment and confrontation promote defensive (fear) responses and escalate aggression
- Management success—combination of environmental control, behavior modification, and medication

Social Status/Dominance Aggression

- Environmental—use barriers and restraint to prevent injury to people
- Devices—train the dog to accept a muzzle and head halter
- Behavior modification, step 1—withdraw all attention from the dog for 2 weeks; list situations in which aggression occurs; devise a method of avoiding each situation; daily, list all aggressive incidents and circumstances to avoid in the future—do not punish the dog
- Behavior modification, step 2—use non-confrontational means to establish the owner's leadership; teach the dog to reliably “sit/stay” on command in gradually more challenging situations (dog must comply without causing any problems to the owner before getting attention and other benefits); no “free” benefits (dog must “sit/stay” before eating, being petted, going for walk, and any other attention; the owner initiates all interactions)
- Behavior modification, step 3—gain greater control; situations that previously elicited aggression are introduced gradually with the dog controlled in a “sit/stay” position (muzzle if necessary)
- Surgery—neuter males; unless aggression is associated with the heat cycle, spaying (ovariohysterectomy) of female will not improve behavior

Inter-male and Inter-female Aggression

- Environmental—use barriers to prevent contact between the dogs, unless they can be well supervised; note dominance order between dogs; if apparent, comply with dogs' rules (for example, dominant dog is fed first, travels through doorways first)
- Devices—train the dog to accept a head halter and muzzle
- A reduced protein diet may be helpful
- Behavior modification, step 1—owner must withdraw all attention to both dogs; teach “sit/stay” program (as for dominance-related aggression)
- Behavior modification, step 2—desensitize or countercondition by gradually decreasing distance between dogs while they are under leash control; reinforce acceptable behavior; “desensitization” is the repeated, controlled exposure to the stimulus [in this case, another dog] that usually causes an aggressive response, in such a way that the dog does not respond with aggression; with repeated efforts, the goal is to decrease the dog's aggressive response; “counterconditioning” is training the dog to perform a positive behavior in place of the negative behavior (in this case aggression)—for example, teaching “sit/stay” and when performed, the dog is rewarded; then when the dog is placed in a situation where it might show aggression, have it “sit/stay”
- Surgery—neuter males; spay (ovariohysterectomy) of females recommended only if aggression is associated with heat cycle (otherwise it will not improve behavior)

Fear-Motivated Aggression

- Environmental—barriers and restraint to prevent injury to people
- Devices—muzzle
- A reduced protein diet may be helpful
- Behavior modification, step 1—list all situations in which the dog appears fearful or exhibits aggression; avoid situations initially; teach dog basic obedience commands and reinforce under non-fearful conditions (generalize by training in many locations)
- Behavior modification, step 2—desensitize and countercondition; subject the dog to mildly fearful conditions with the stimulus (for

example, a stranger) far away; keep the dog attentive and performing obedience commands; gradually decrease the distance of the stranger; if the dog exhibits fear, the stranger should withdraw and work should continue at an easier level, then gradually progress; desensitize or countercondition by gradually decreasing distance between the dog and the stimulus while the dog is under leash control; reinforce acceptable behavior; “desensitization” is the repeated, controlled exposure to the stimulus that usually causes an aggressive response, in such a way that the dog does not respond with aggression; with repeated efforts, the goal is to decrease the dog’s aggressive response; “counterconditioning” is training the dog to perform a positive behavior in place of the negative behavior (in this case aggression)—for example, teaching “sit/stay” and when performed, the dog is rewarded; then when the dog is placed in a situation where it might show aggression, have it “sit/stay”

- Surgery—neutering males or spaying female probably will not improve the behavior

Territorial Aggression

- Environmental—barriers and restraint to prevent injury to people; initially, when visitors come, isolate the dog to prevent it from exhibiting the behavior
- Devices—head halter, muzzle
- Behavior modification, step 1—teach the dog “sit/stay,” first at neutral locations, then near the door and at other sites of territorial aggression; later, control the dog while a familiar person approaches; reward the dog for calm, obedient behavior
- Behavior modification, step 2—gradually introduce strangers; increase the difficulty as the dog learns control; move the exercises to the door; add ringing the door bell and entering the door
- Surgery—neutering males or spaying female probably will not improve the behavior

DIET

- A reduced protein diet may be helpful in controlling some forms of aggression



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No medications are approved by the federal Food and Drug Administration (FDA) for the treatment of aggression in dogs; discuss the risks and benefits of using medications with your pet’s veterinarian
- Medication should be used only in conjunction with a safe management plan
- Medications that increase serotonin (chemical messenger in the brain that affects mood and behavior) may be helpful to reduce anxiety, arousal, and impulsivity
- Treatment duration: 4 months to life
- Medications that have been tried include amitriptyline, fluoxetine, and L-tryptophan
- Clomipramine (Clomicalm®) has a warning on the label that it is not designed to treat aggression; therefore, it should not be used to treat canine aggression
- Megestrol acetate has been used successfully with dominance-related and inter-male aggression; however, it does have side effects that should be considered



FOLLOW-UP CARE

PATIENT MONITORING

- Weekly to biweekly contact—recommended in the initial phases
- Clients need feedback and assistance with behavior modification plans and medication management

PREVENTIONS AND AVOIDANCE

- Avoid situations that lead to aggression
- Use extreme care when handling aggressive dogs; use muzzles and other restraints to prevent injury to people and other animals

POSSIBLE COMPLICATIONS

- Injury to people and/or other animals
- Social status/dominance aggression—can be directed toward owners
- Interdog aggression—people often seriously injured when interfering with fighting dogs, either by accident or by redirected or irritable aggression; owners should not reach for fighting dogs; pull apart with leashes

EXPECTED COURSE AND PROGNOSIS

- Aggressive dogs are never cured, some may be managed successfully



KEY POINTS

- Aggressive dogs are never cured, some may be managed successfully
- Behavioral warning signs include being motionless (immobility), growling, snarling, or snapping at air; offensive aggression warning signs include head up, tail up, direct stare, face-on immobility; defensive aggression warning signs include head lowered, tail down, and body withdrawn
- Avoid situations that lead to aggression
- Use extreme care when handling aggressive dogs; use muzzles and other restraints to prevent injury to people and other animals

HAIR LOSS (ALOPECIA) IN CATS



BASICS

OVERVIEW

- “Alopecia” is the medical term for hair loss
- Hair loss is a common problem in cats
- Characterized by a complete or partial lack of hair in areas where it is present normally
- Pattern of hair loss—varied or symmetrical

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Mean Age and Range

- Cancer-related hair loss (alopecia)—generally recognized in old cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Hair loss; pattern of hair loss varies—may be localized or widespread
- Skin itself may appear normal or may be abnormal (such as redness; multiple, pinpoint bumps or scabs; or loss of superficial layers of the skin [known as ulceration])
- Other signs depend on the underlying cause of hair loss

CAUSES

- Nervous system or behavioral disorders—obsessive-compulsive disorder, in which the cat over grooms, with resulting hair loss
- Hormonal disorders—sex hormone hair loss (alopecia); excessive levels of thyroid hormone (known as “hyperthyroidism”); increased levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); diabetes mellitus (“sugar diabetes”)
- Immune-mediated disorders—skin allergies (known as “allergic dermatitis”); specific condition characterized by multiple patches of hair loss (known as “alopecia areata”)
- Parasites—demodectic mange (known as “demodicosis”)
- Fungal infection—ringworm (known as “dermatophytosis”)
- Physiologic disorder—condition characterized by multiple areas of hair loss with reddened skin, scales (accumulations of surface skin cells, such as seen in dandruff), and signs of itchiness (known as “pruritus”) with inflammation of the sebaceous glands, the glands that produce oils in the hair coat (condition known as “sebaceous adenitis”)
- Cancer or cancer-related hair loss
- Unknown cause (so called “idiopathic disease”)
- Inherited hair loss

RISK FACTORS

- Feline leukemia virus (FeLV) infection and feline immunodeficiency virus (FIV) infection—for demodectic mange (demodicosis)



TREATMENT

HEALTH CARE

- Treatment is limited for many of the disorders that cause hair loss (alopecia)
- Behavioral modification or use of a “T-shirt” on the cat may help prevent excessive self-grooming
- Shampoo and treatment applied directly to the skin may help secondary problems, such as increased thickness of the outer, keratinized layer of the skin (known as “hyperkeratosis”) in sebaceous adenitis (condition with hair loss, reddened skin, scales and inflammation of the oil-secreting sebaceous glands); dried discharge on the surface of the skin lesion (known as a “crust”) in demodectic mange (demodicosis); secondary bacterial infections; and malodor for greasy conditions

DIET

- Removal of an offending dietary item may alleviate signs of food allergy (such as hair loss, scratching at skin)

SURGERY

- Biopsy of a tumor or the skin may be indicated in the diagnostic work-up for some causes of hair loss (alopecia)
- Excessive levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing’s disease)—surgical removal of the adrenal gland

- Surgical removal of skin cancer or tumors



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Obsessive-compulsive disorder—amitriptyline
- Hormonal hair loss (alopecia) in males—testosterone supplementation
- Skin allergy (allergic dermatitis)—antihistamines, steroids, “allergy shots” (known as “hyposensitization vaccine”)
- Excessive levels of thyroid hormone (hyperthyroidism)—medications given by mouth, such as methimazole (Tapazole®), or radioactive iodine therapy
- Diabetes mellitus (“sugar diabetes”)—regulation of glucose levels with insulin
- Excessive levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing’s disease)—surgery; no known effective medical therapy
- Cancer-related hair loss (alopecia)—no therapy for many types of cancer-related hair loss; disease often fatal
- Epidermotropic lymphoma (type of cancer in the skin characterized by the presence of abnormal lymphocytes; a lymphocyte is a type of white-blood cell, formed in lymphatic tissue throughout the body)—retinoids (isotretinoin), steroids, interferon, cyclosporine, lomustine
- Sebaceous adenitis (condition with hair loss, reddened skin, scales and inflammation of the oil-secreting sebaceous glands)—retinoids, steroids, cyclosporine
- Squamous cell carcinoma (type of skin cancer)—retinoids (applied to skin directly [topical] and administered by mouth [oral]), topical imiquimod cream
- Alopecia areata (specific condition involving multiple patches of hair loss)—no therapy; possibly counterirritants
- Demodectic mange (demodicosis)—lime sulfur dips at weekly intervals for 4 to 6 dips; Mitaban® and ivermectin have been tried with variable success
- Ringworm (dermatophytosis)—griseofulvin, ketoconazole, itraconazole (best choice), lufenuron



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on specific diagnosis

PREVENTIONS AND AVOIDANCE

- Depend on specific diagnosis

POSSIBLE COMPLICATIONS

- Depend on specific diagnosis

EXPECTED COURSE AND PROGNOSIS

- Depend on specific diagnosis



KEY POINTS

- “Alopecia” is the medical term for hair loss
- Hair loss is a common problem in cats
- Pattern of hair loss varies—may be localized or widespread
- Skin itself may appear normal or may be abnormal

HAIR LOSS (ALOPECIA) IN DOGS



BASICS

OVERVIEW

- “Alopecia” is the medical term for hair loss
- Hair loss is a common disorder in dogs
- Characterized by a complete or partial lack of hair in areas where it is present normally
- Pattern of hair loss—varied or symmetrical
- May be the primary problem or a secondary phenomenon

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- May be sudden (acute) in onset or slowly progressive
- Multiple patches of circular hair loss (alopecia)—most frequently associated with inflammation of the hair follicles (known as “folliculitis”) from bacterial infection and/or demodectic mange (known as “demodicosis”)
- Large, more widespread areas of hair loss (alopecia)—may indicate abnormal development of the hair follicles or hair (known as “follicular dysplasia”) or a more generalized disease
- The pattern and degree of hair loss are important for establishing a diagnosis

CAUSES

Multiple Areas (Multifocal) of Hair Loss

- Localized demodectic mange (demodicosis)—partial to complete hair loss (alopecia) with reddening of the skin (known as “erythema”) and mild scaling; lesions may become inflamed and may have dried discharge on the surface (dried discharge known as “crusts”)
- Ringworm (known as “dermatophytosis”)—“ringworm” is a fungal infection on the surface of the skin characterized by partial to complete hair loss (alopecia) with scaling; with or without reddening of the skin (erythema); not always “ring-like” in appearance
- Inflammation of the hair follicles due to *Staphylococcus* bacterial infection (known as “staphylococcal folliculitis”)—circular patterns of hair loss (alopecia) bordered by scales (accumulations of surface skin cells, such as seen in dandruff) or surface peeling of the skin (the pattern is known as an “epidermal collarette”), reddening of the skin (erythema), dried discharge on the surface of the skin lesion (crust), and darkened areas of skin (known as “hyperpigmented macules”)
- Injection reactions—inflammation with hair loss (alopecia) and/or thinning of the skin (known as “cutaneous atrophy”) from scarring
- Rabies-vaccine inflammation of the blood vessels (known as “vasculitis”)—patch of hair loss (alopecia) at the location where the rabies vaccine was administered is observed 2 to 3 months following vaccination
- Localized scleroderma (condition in which normal skin is replaced by scar tissue for some unknown cause)—well-demarcated, shiny, smooth skin with hair loss (alopecia); lesion is a thickened, raised, flat-topped area that is slightly higher than the normal skin (known as a “plaque”)
- Specific condition characterized by multiple patches of hair loss (known as “alopecia areata”)—noninflammatory areas of complete hair loss (alopecia)
- Condition characterized by multiple areas of hair loss with reddened skin, scales, and signs of itchiness (known as “pruritus”) with inflammation of the sebaceous glands, the glands that produce oils in the hair coat (condition known as “sebaceous adenitis”) seen in short-coated breeds—ring-like areas of hair loss (alopecia) and scaling

Symmetrical Hair Loss

- Excessive levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)—hair loss along the sides of the body (known as “truncal alopecia”) associated with thin skin, plugs of keratin and oil in the follicles of the skin (known as “comedones”), and skin infection characterized by the presence of pus (known as “pyoderma”)
- Inadequate levels of thyroid hormone (known as “hypothyroidism”)—hair loss (alopecia) is an uncommon presentation
- Growth hormone-responsive skin disorder (known as “growth hormone-responsive dermatosis”)—symmetrical hair loss along the sides of the body (truncal alopecia) associated with darkened skin (known as “hyperpigmentation”); hair loss often starts along the collar area of the neck
- Excessive levels of estrogen (known as “hyperestrogenism”) in females—symmetrical hair loss (alopecia) of the flanks and skin between the external genitalia and the anus (perineal skin) and between the rear legs (inguinal skin) with enlarged external genitalia (vulva) and mammary glands
- Inadequate secretion of female hormones (known as “hypogonadism”) in intact females—hair loss of the skin between the external genitalia and the anus (perineal skin), flank, and hair loss along the sides of the body (truncal alopecia)
- Testosterone-responsive skin disorder (known as “testosterone-responsive dermatosis”) in castrated males—slowly progressive hair loss along the sides of the body (truncal alopecia)
- Male feminization from Sertoli cell tumor (a type of tumor in the testicles)—hair loss (alopecia) of the skin between the external genitalia and the anus (perineal skin) and genital region with excessive development of the male mammary glands (known as “gynecomastia”)

- Castration-responsive skin disorder (known as “castration-responsive dermatosis”)—hair loss (alopecia) in the collar area, rump, skin between the external genitalia and the anus (perineal skin), and flanks
- Estrogen-responsive skin disorder (known as “estrogen-responsive dermatosis”) in spayed female dogs—hair loss (alopecia) of the skin between the external genitalia and the anus (perineal skin) and genital regions
- Seasonal flank hair loss (alopecia)—creeping hair loss involving the flanks with darkened skin (hyperpigmentation)

Patchy to Generalized (Diffuse) Hair Loss

- Demodectic mange (demodicosis)—often associated with reddening of the skin (erythema), inflammation of the hair follicles (folliculitis), and darkened skin (hyperpigmentation)
- Bacterial infection/inflammation of the hair follicles (folliculitis)—multiple areas of circular hair loss (alopecia) that may join to form large areas of hair loss; circular patterns of hair loss bordered by scales (accumulations of surface skin cells, such as seen in dandruff) or surface peeling of the skin (epidermal collarette)
- Ringworm (dermatophytosis)—often accompanied by scales (accumulations of surface skin cells, such as seen in dandruff)
- Sebaceous adenitis (condition characterized by multiple areas of hair loss with reddened skin, scales, and signs of itchiness [known as “pruritus”] with inflammation of the sebaceous glands, the glands that produce oils in the hair coat)—hair loss (alopecia) with thick, adherent scales; predominantly along the back line of the body, including the head
- Color-mutant hair loss (alopecia)—thinning of the hair coat with secondary inflammation of the hair follicles (folliculitis) in some blue or fawn dogs
- Abnormal development of the hair follicles or hair (known as “follicular dysplasia”)—slowly progressive hair loss (alopecia)
- Hair loss during stages of the hair growth cycle—sudden (acute) onset of hair loss (alopecia)
- Inadequate levels of thyroid hormone (hypothyroidism)—generalized (diffuse) thinning of the hair coat
- Excessive levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing’s disease)—hair loss along the sides of the body (truncal alopecia) with thin skin and formation of plugs of keratin and oil in the follicles of the skin (comedones)
- Epidermotropic lymphoma (type of cancer in the skin characterized by the presence of abnormal lymphocytes; a lymphocyte is a type of white-blood cell, formed in lymphatic tissue throughout the body)—widespread, generalized hair loss along the sides of the body (truncal alopecia) with scales (accumulations of surface skin cells, such as seen in dandruff) and reddening of the skin (erythema); later small, solid masses (known as “nodules”) and thickened, raised, flat-topped areas that are slightly higher than the normal skin (known as “plaques”) may form
- Pemphigus foliaceus (a disease in which the body’s immune system attacks its own skin)—hair loss (alopecia) associated with the formation of scales (accumulations of surface skin cells, such as seen in dandruff) and dried discharge on the skin lesions (crusts)
- Keratinization disorders (disorders in which the surface of the skin is abnormal)—hair loss (alopecia) associated with excessive scales (accumulations of surface skin cells, such as seen in dandruff) and greasy surface texture

Specific Locations of Hair Loss

- Hair loss involving the ears (known as “pinna alopecia”)—miniaturization of hairs and progressive hair loss (alopecia)
- Traction hair loss (alopecia)—hair loss on the top and sides of the head secondary to having barrettes or rubber bands applied to the hair
- Postclipping hair loss (alopecia)—failure to regrow hair after clipping
- Melanoderma (hair loss [alopecia] of Yorkshire terriers)—symmetrical hair loss with darkened skin of the ears, bridge of the nose, tail, and feet
- Seasonal flank hair loss (alopecia)—creeping hair loss of the flanks, that may connect over the back
- Abnormal development of the hair follicles or hair involving black hairs only (known as “black hair follicular dysplasia”)—hair loss (alopecia) involving only the black-haired areas of the body
- Inherited inflammatory disorder that affects the skin and muscles of unknown cause (condition known as “idiopathic familial canine dermatomyositis”) in collies and Shetland sheepdogs—hair loss (alopecia) of the face, tip of ears, tail, and digits; associated with scales (accumulations of surface skin cells, such as seen in dandruff) and dried discharge on the skin lesions (crusts), and scarring



TREATMENT

HEALTH CARE

- Demodectic mange (demodicosis)—amitraz, ivermectin, Interceptor®
- Ringworm (dermatophytosis)—griseofulvin, ketoconazole, itraconazole, lime sulfur dips, lufenuron
- Inflammation of hair follicles due to *Staphylococcus* bacterial infection (staphylococcal folliculitis)—shampoo and antibiotic therapy
- Sebaceous adenitis (condition with hair loss, reddened skin, scales and inflammation of the oil-secreting sebaceous glands)—keratolytic shampoo, essential fatty acid supplementation, retinoids
- Keratinization disorders (disorders in which the surface of the skin is abnormal)—shampoos, retinoids, vitamin D

SURGERY

- Biopsy of a tumor or the skin may be indicated in the diagnostic work-up for some causes of hair loss (alopecia)
- Hormonal disorders causing hair loss (treatment determined by specific hormonal disorder)—surgery may include removal of ovaries and uterus (known as “ovariohysterectomy” or “spay”), removal of testicles (known as “castration”), or removal of adrenal glands (known as “adrenalectomy”)
- Surgical removal of skin cancer or tumors



MEDICATIONS

- Vary with specific cause
- Excessive levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing's disease)—Lysodren®



FOLLOW-UP CARE

PATIENT MONITORING

- Varies with specific cause

PREVENTIONS AND AVOIDANCE

- Vary with specific cause

POSSIBLE COMPLICATIONS

- Vary with specific cause

EXPECTED COURSE AND PROGNOSIS

- Vary with specific cause



KEY POINTS

- “Alopecia” is the medical term for hair loss
- Hair loss is a common problem in dogs
- Pattern of hair loss varies—may be localized or widespread
- Skin itself may appear normal or may be abnormal

AMYLOIDOSIS

(DISORDER CAUSED BY DEPOSITION OF PROTEINS [AMYLOID] IN VARIOUS ORGANS)



BASICS

OVERVIEW

- A group of conditions of differing cause in which insoluble proteins (amyloid) are deposited outside the cells in various tissues and organs, compromising the normal function of the tissues or organs

GENETICS

- No genetic involvement is established clearly; occurs in certain lines or families (known as “familial amyloidosis”) in the following dog breeds: Chinese shar pei, English foxhound, and beagle, and in the following cat breeds: Abyssinian, Oriental shorthair, and Siamese

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Uncommon disease in domestic animals; occurs most commonly in dogs; rare in cats, except Abyssinians

Breed Predilections

- Dogs—Chinese shar pei, beagle, collie, pointer, English foxhound, and walker hound; German shepherd dog and mixed-breed dogs are at lower risk
- Cats: Abyssinian, Oriental shorthair, and Siamese

Mean Age and Range

- Most affected dogs and cats are older than 5 years of age
- Dogs—mean age at diagnosis is 9 years; range, 1–15 years
- Cats—mean age at diagnosis is 7 years; range, 1–17 years
- Prevalence increases with age
- Abyssinian cats—range, less than 1 year of age and up to 17 years of age
- Chinese shar pei—usually less than 6 years of age when signs of kidney failure develop; range, 1.5 to 6 years of age
- Siamese cats with familial amyloidosis of the liver and thyroid gland usually develop signs of liver disease when 1 to 4 years of age

Predominant Sex

- Dogs and Abyssinian cats—females appear to be at a slightly higher risk than males to develop amyloidosis

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on the organs affected, the amount of amyloid present in the tissues or organs, and the reaction of the affected tissues and organs to amyloid deposits
- Signs usually caused by kidney involvement; occasionally, liver involvement may cause signs in Chinese shar pei dogs and Oriental shorthair and Siamese cats
- Lack of appetite (anorexia), sluggishness (lethargy), excessive urination (polyuria) and excessive thirst (polydipsia), weight loss, vomiting, and occasionally diarrhea
- Fluid build-up in the abdomen (known as “ascites”) and fluid build-up under the skin in the limbs and other parts of the body (known as “peripheral edema”) may be seen in animals with nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin)
- Chinese shar pei may have a history of previous episodic joint swelling and high fever that resolved spontaneously within a few days
- Young beagles with inflammation of many arteries (known as “juvenile polyarteritis”) may have a history of fever and neck pain that persisted for 3–7 days
- Oriental shorthair and Siamese cats may present with spontaneous bleeding in the liver, leading to acute collapse and accumulation of blood in the abdomen (known as “hemoabdomen”)
- Signs related to kidney failure—ulcers in the mouth, extreme weight loss (emaciation), vomiting, and dehydration; on physical examination, kidneys may be small, normal-sized, or slightly enlarged in affected dogs; they are usually small, firm, and irregular in affected cats
- Signs related to the primary inflammatory disease or cancer that caused the build-up of the amyloid protein in the tissues
- Up to 40% of affected dogs may develop blockage of blood vessels due to the presence of blood clots (thromboembolic phenomena); signs vary with the location of the blood clot (thrombus); patients may develop difficulty breathing (dyspnea) if the clot forms in or moves into the lungs (known as “pulmonary thromboembolism”) or may develop weakness or paralysis of one or both hind limbs if the clot is located in the arteries going to the hind limbs (known as “iliac or femoral artery thromboembolism”)
- Chinese shar pei dogs and Oriental shorthair and Siamese cats may have signs of liver disease (such as yellowish discoloration to the tissues [jaundice or icterus], wasting with extreme weight loss [cachexia], and spontaneous liver rupture and internal bleeding)

CAUSES

- Chronic inflammation—systemic fungal infections (known as “mycoses,” such as blastomycosis, coccidioidomycosis); chronic bacterial infections (such as infections of the bone [osteomyelitis], of the bronchi and lungs [bronchopneumonia], inflammation of the lining of the chest [pleuritis], inflammation of the fat [steatitis], inflammation/infection of the uterus [pyometra], inflammation/infection of the kidney [pyelonephritis], chronic skin inflammation with pus present [suppurative dermatitis], chronic joint inflammation with pus present [suppurative arthritis], chronic inflammation of the lining of the abdomen [peritonitis], chronic inflammation of the mouth [stomatitis]); parasitic infections (such as heartworm disease [dirofilariasis], leishmaniasis, hepatozoonosis); and immune-mediated diseases (such as systemic lupus erythematosus)
- Cancer (examples include lymphoma, plasmacytoma, multiple myeloma, mammary tumors, testicular tumors)
- Familial (seen in Chinese shar pei, English foxhound and beagle dogs; Abyssinian, Siamese, and Oriental shorthair cats)
- Others— inherited disease in gray collies in which the dog has repeated episodes of low white-blood cell counts and fever (known as “cyclic hematopoiesis”); disease in young beagles with inflammation of many arteries (juvenile polyarteritis)

RISK FACTORS

- Chronic inflammation or cancer
- Family history in certain breeds



TREATMENT

HEALTH CARE

- Hospitalize patients with chronic kidney failure and dehydration for initial medical management
- Can manage stable patients and those that have protein in the urine, but no clinical signs (known as “asymptomatic proteinuria”) as outpatients
- Correct dehydration with 0.9% NaCl (sodium chloride) solution or lactated Ringer’s solution; patients with severe metabolic acidosis (a condition in which levels of acid are increased in the blood) may benefit from bicarbonate supplementation
- Identify underlying inflammatory conditions or cancer and treat, if possible
- Manage kidney failure

ACTIVITY

- Normal

DIET

- Patients with chronic kidney failure—restrict phosphorus and moderately restrict protein
- Patients with high blood pressure (hypertension)—restrict sodium



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medication to control blood pressure in patients with high blood pressure (hypertension)
- Patients with blood clots (thromboembolic syndrome) and nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin) caused by accumulation of amyloid protein in the glomerulus of the kidney (known as “glomerular amyloidosis”) usually have a low plasma concentration of antithrombin III (a compound involved in clotting of the blood); low-dose aspirin has been suggested for dogs with glomerular disease to prevent platelet aggregation (treatment with aspirin should only be under the supervision of your pet’s veterinarian)
- DMSO—may be helpful
- Methylsulfonylmethane (MSM) has been used in dogs with amyloidosis, but no evidence indicates that it benefits dogs with kidney amyloidosis
- Colchicine—prevents development of amyloidosis in humans with familial Mediterranean fever (a familial amyloidosis) and stabilizes kidney function in patients with nephrotic syndrome, but without signs of kidney failure; no evidence of benefit once the patient develops kidney failure; may cause vomiting, diarrhea, and low white blood cell counts (neutropenia) in dogs; colchicine is used particularly in the Chinese shar pei with episodic fever or multi-joint arthritis (polyarthritis) before development of kidney failure



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor appetite and activity level daily; check body weight weekly
- Serum blood tests, especially albumin, creatinine, and blood urea nitrogen (BUN) concentrations, every 2–6 months in stable patients
- Can assess degree of protein being lost in the urine (proteinuria) by repeated urine protein: creatinine (UP/C) ratios

PREVENTIONS AND AVOIDANCE

- Do not breed affected animals

POSSIBLE COMPLICATIONS

- Kidney failure
- Nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin)
- Systemic high blood pressure (hypertension)
- Liver rupture, causing bleeding into the abdomen
- Blood clots (thromboembolic disease)

EXPECTED COURSE AND PROGNOSIS

- Progressive disease that is usually advanced at the time of diagnosis; prognosis improves if an underlying immune-mediated disease, inflammatory disease or cancer is detected and treated successfully
- Survival for dogs with glomerular amyloidosis varied from 3 to 20 months in one study; some dogs occasionally may live longer
- Cats with kidney failure because of amyloidosis usually survive less than 1 year
- Mildly affected cats may not develop kidney failure and have an almost a normal life expectancy



KEY POINTS

- Progressive disease that is usually advanced at the time of diagnosis; prognosis improves if an underlying immune-mediated disease, inflammatory disease or cancer is detected and treated successfully
- Signs usually caused by kidney involvement; occasionally, liver involvement may cause signs in Chinese shar pei dogs and Oriental shorthair and Siamese cats
- Familial predisposition in susceptible breeds; familial amyloidosis occurs in the following dog breeds: Chinese shar pei, English foxhound, and beagle, and in the following cat breeds: Abyssinian, Oriental shorthair, and Siamese
- Potential for complications (such as high blood pressure and blood clots)

ANAL SAC/PERIANAL ADENOCARCINOMA

(CANCER INVOLVING THE ANAL SAC OR AREA AROUND THE ANUS)



BASICS

OVERVIEW

- Uncommon cancerous tumor (malignant neoplasm) that developed from glands of the anal sac
- Locally spreading (invasive) cancer
- High rate of spread to other areas of the body (metastasis), often to the lymph nodes under the lumbar spine (sublumbar lymph nodes)
- Frequently associated with high blood calcium levels (hypercalcemia)

SIGNALMENT/DESCRIPTION of ANIMAL

- Older dogs; extremely rare in cats
- Females have had higher rates of anal sac/perianal adenocarcinoma in some studies
- No breed has been proven to have increased likelihood of developing anal sac/perianal adenocarcinoma

SIGNS/OBSERVED CHANGES in the ANIMAL

- Mass associated with anal sac, straining to defecate, and/or constipation
- May have lack of appetite (anorexia), excessive thirst (polydipsia), excessive urination (polyuria), and sluggishness (lethargy)
- Mass associated with anal sac may be quite small despite massive metastatic disease

CAUSES

- A hormonal cause is hypothesized



TREATMENT

HEALTH CARE

- Surgery is the treatment of choice for the primary tumor
- Surgical removal of the primary tumor and enlarged lymph nodes may prolong survival
- Radiation may be helpful, but acute and chronic radiation side effects can be moderate to severe
- Consult a veterinary oncologist for current recommendations
- Monitor blood calcium levels and manage high levels (hypercalcemia), if present

DIET

- Normal diet or as recommended by your pet's veterinarian

SURGERY

- Surgical removal (resection) of the tumor
- Partial surgical removal to decrease the size (debulking) of the tumor in cases where the tumor cannot be totally removed
- Surgical removal or decrease in size of lymph nodes with evidence of metastasis



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Limited reports of partial responses to platinum-containing chemotherapeutic compounds in dogs—cisplatin, carboplatin
- Possible role for melphalan after debulking surgery



FOLLOW-UP CARE

PATIENT MONITORING

- Complete surgical tumor removal—physical examination, chest X-rays, abdominal ultrasonography, and blood work (serum biochemistry tests) as scheduled by your pet's veterinarian
- Partial surgical tumor removal—monitor tumor size and blood calcium levels and kidney tests (blood work, urinalysis)

EXPECTED COURSE AND PROGNOSIS

- Prognosis guarded to poor
- Surgery often reduces the severity of signs (known as “palliative” treatment), but is not curative
- May see both local progression of the tumor and metastasis occurring
- Growth of the tumor may be slow and debulking lymph-node metastatic disease may significantly prolong survival
- Presence of high blood calcium levels and metastasis were poor prognostic factors in one study
- Median survival time (the time between diagnosis and death) ranges from about 8 to 19 months, depending on individual clinical status
- Ultimately, dogs succumb to complications related to high blood calcium levels or from effects of the primary tumor or metastases



KEY POINTS

- Uncommon cancerous tumor (malignant neoplasm) that developed from glands of the anal sac
- High rate of spread to other areas of the body (metastasis)
- Frequently associated with high blood calcium levels (hypercalcemia)
- Surgical removal of the primary tumor and enlarged lymph nodes may prolong survival
- Prognosis guarded to poor

ANAPHYLAXIS



BASICS

OVERVIEW

- “Allergy” is an unusual sensitivity to a substance (such as pollen)—the immune system responds to the presence of the substance leading to signs (such as itchiness); “antigen” is a substance (such as pollen) that induces a sensitivity or immune response; “antibody” is a protein that is produced by the immune system in response to a specific antigen—when the body is exposed to the antigen, the antibody responds, causing the signs of the allergic response
- “Immunoglobulins” are proteins produced by the cells of the immune system; they include the antibodies; they are categorized into classes, including immunoglobulin A (IgA), immunoglobulin G (IgG), and immunoglobulin E (IgE)
- Mast cells are immune-system cells that frequently are located near blood vessels in the skin; mast cells contain histamine; they are involved in allergy and inflammation
- Anaphylaxis is the sudden (acute) allergic reaction following the rapid introduction of an antigen (a substance that induces sensitivity or immune response) into a host having antigen-specific antibodies (proteins produced by the immune system in response to a specific antigen) of the immunoglobulin E (IgE) subclass
- The binding of antigen (a substance that induces sensitivity or immune response) to mast cells sensitized with immunoglobulin E (IgE) results in the release of preformed and newly synthesized chemical mediators (such as histamine)
- Anaphylactic reactions may be localized (atopy) or generalized (systemic), known as “anaphylactic shock”
- “Atopy” is a disease in which the animal is sensitized (or “allergic”) to substances found in the environment (such as pollen) that normally would not cause any health problems
- “Anaphylactic shock” is a severe form of anaphylaxis; it is life-threatening; signs may include difficulty breathing, vomiting, diarrhea, collapse, and death

GENETICS

- Anaphylaxis may be more common in some families or lines of dogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dogs—numerous breeds documented as being susceptible for developing atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems)
- Cats—no breeds documented as having a susceptibility for atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems)

Mean Age and Range

- Dogs—age of onset of signs ranges from 3 months to several years of age; most affected animals are 1 to 3 years of age when signs are first identified
- Cats—age of onset of signs ranges from 6 months to 2 years

Predominant Sex

- Dogs—atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems) more common in females
- Cats—no reported differences between males and females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Initial clinical signs vary depending on the route of exposure (such as airborne, injection) to the antigen (the substance [such as a vaccine] that induces a sensitivity or immune response) that is causing the reaction
- Shock—end result of a severe anaphylactic reaction
- Shock organ—dogs, liver; cats, respiratory and gastrointestinal systems
- May be localized to the site of exposure, but may progress to a generalized (systemic) reaction
- Onset of signs immediate (usually within minutes of exposure to substance that induces the allergic response)
- Dogs—itchiness (known as “pruritus”); hives; vomiting; defecation; and urination
- Cats—intense itchiness (pruritus) about the head; difficulty breathing (known as “dyspnea”); salivation; and vomiting
- Localized fluid build-up in the skin (known as “cutaneous edema”) at the site of exposure (such as at the site of an insect sting or an injection)
- Enlarged liver (known as “hepatomegaly”) in some dogs
- Increased excitement possible in early stages
- Depression and collapse terminally

CAUSES

- Virtually any substance; those commonly reported include venoms, blood-based products, vaccines, foods, and drugs

RISK FACTORS

- Previous exposure (sensitization) increases the chance of the animal developing a reaction



TREATMENT

HEALTH CARE

- In a suddenly affected animal, the reaction is considered a medical emergency requiring hospitalization
- Eliminate the antigen (the substance [such as a vaccine] that induces a sensitivity or immune response) that is causing the reaction, if possible

Generalized (Systemic) Anaphylaxis

- Goal—emergency life support through the maintenance of an open airway, preventing circulatory collapse, and re-establishing normal body function
- Administer fluids intravenously at shock dosages to counteract low blood pressure (known as “hypotension”)

Localized Anaphylaxis

- Goal—limit the reaction and prevent progression to a generalized (systemic) reaction

DIET

- If a food is suspected as the cause of the anaphylaxis (uncommon), avoid foods associated with allergic reaction



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Generalized (Systemic) Anaphylaxis

- Epinephrine for shock (administered by injection)
- Steroids for shock—prednisolone or dexamethasone (administered by injection)
- Atropine to counteract slow heart rate (known as “bradycardia”) and low blood pressure (hypotension)
- Aminophylline is a drug that enlarges the bronchi and bronchioles in the lungs (class of drugs known as “bronchodilators”); can be used in patients having severe breathing difficulties

Localized Anaphylaxis

- Diphenhydramine (administered by injection) is an antihistamine
- Prednisolone
- Epinephrine (administered by injection, at site of initiation)
- If shock develops, initiate treatment for generalized (systemic) anaphylaxis



FOLLOW-UP CARE

PATIENT MONITORING

- Closely monitor hospitalized patients for 24 to 48 hours

PREVENTIONS AND AVOIDANCE

- If antigen (the substance [such as a vaccine] that induces a sensitivity or immune response) that caused anaphylaxis can be identified, eliminate or reduce exposure

POSSIBLE COMPLICATIONS

- Death

EXPECTED COURSE AND PROGNOSIS

- If localized reaction is treated early, prognosis is good
- If the animal is in shock on examination, prognosis is guarded to poor



KEY POINTS

- Anaphylaxis is an unpredictable disease
- Recognize that the animal has an allergic condition and may require immediate medical care
- In a suddenly affected animal, the reaction is considered a medical emergency
- Eliminate the antigen (the substance [such as a vaccine] that induces a sensitivity or immune response) that is causing the reaction, if possible

IMMUNE-MEDIATED ANEMIA

(DESTRUCTION OF RED-BLOOD CELLS CAUSED BY AN IMMUNE RESPONSE)



BASICS

OVERVIEW

- Accelerated destruction or removal of red-blood cells related to an immune response, in which the body produces antibodies against red-blood cells
- Also known as “immune-mediated hemolytic anemia” or “IMHA”
- “Anemia” is a low red-blood cell count; “hemolytic” refers to hemolysis; “hemolysis” is the destruction of red-blood cells, which allows the release of hemoglobin (the compound in the red-blood cells that carries oxygen to the tissues of the body)
- “Antibody” is a protein that is produced by the immune system in response to a specific antigen (in this case, on the red-blood cells); two groups of antibodies have been identified that are involved in immune-mediated anemia, based on characteristics identified in the laboratory: warm-reacting antibodies and cold-reacting antibodies

GENETICS

- Isolated families of dogs have been documented to be affected (breeds include the Old English sheepdog, Vizsla, Scottish terrier, cocker spaniel, and miniature schnauzer)
- No genetic basis has been established

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Cats

Breed Predilections

- Dog breeds—Old English sheepdog, Vizsla, Scottish terrier, cocker spaniel, and miniature schnauzer; other commonly affected dog breeds include the miniature poodle, Irish setter, English springer spaniel, Doberman pinscher, and collie
- Cat breed—domestic shorthair

Mean Age and Range

- In dogs, mean age, 5 to 6 years; reported range of 1 to 13 years of age
- In cats, mean age, 3 years; reported range of 0.5 to 9 years of age

Predominant Sex

- Females may have a higher risk than males in dogs
- Males have a higher risk than females in cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Collapse
- Weakness
- Sluggishness (lethargy)
- Lack of appetite (known as “anorexia”)
- Eating of nonfood items (known as “pica”) in cats
- Exercise intolerance
- Difficulty breathing (known as “dyspnea”)
- Rapid breathing (known as “tachypnea”)
- Vomiting
- Diarrhea
- Occasionally increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)
- Pale gums and moist tissues of the body (tissues known as “mucous membranes”); rapid heart rate (known as “tachycardia”)
- Enlarged spleen (known as “splenomegaly”) and enlarged liver (known as “hepatomegaly”)
- Yellowish discoloration to gums and moist tissues of the body (known as “icterus” or “jaundice”) and dark urine (known as “pigmenturia”) due to the presence of hemoglobin (a breakdown product of red-blood cells) or bilirubin (a bile pigment that is in increased levels with icterus)
- Fever and enlarged lymph nodes (known as “lymphadenopathy”)
- Heart murmur and “gallop” sound on listening to the heart with a stethoscope
- Bruising or dark, tarry stools due to the presence of digested blood (condition known as “melena”) possible in animals with coexistent low platelet counts (known as “thrombocytopenia”) or a blood-clotting disorder (“disseminated intravascular coagulopathy” or “DIC”)
- Skin lesions may be seen
- Other generalized (systemic) signs possible (such as joint pain and kidney disease) if immune-mediated hemolytic anemia (IMHA) is a component of systemic lupus erythematosus (autoimmune disease in which body attacks its own skin and other organs)

CAUSES

Primary Immune-Mediated Hemolytic Anemia (IMHA)

- Autoimmune hemolytic anemia, where the body's immune system attacks its own red-blood cells
- Systemic lupus erythematosus (autoimmune disease in which body attacks its own skin and other organs)
- Breakdown of red-blood cells due to the presence of antibodies from the mother in the milk (condition known as “neonatal isoerythrolysis”)—queen (mother cat) with blood type B; kitten with blood type A
- Abnormal immune system
- Infectious agents (such as bacteria and viruses) and medications
- Unknown cause (known as “idiopathic immune-mediated anemia”)

Secondary Immune-Mediated Hemolytic Anemia (IMHA)

- Infectious causes (such as *Mycoplasma*, *Babesia*, *Leptospira*, *Ehrlichia*, feline leukemia virus [FeLV], feline infectious peritonitis [FIP])
- Heartworm disease
- Cancer
- Medications (such as cephalosporins, propylthiouracil, and methimazole)

RISK FACTORS

- Exposure to infectious agents, vaccination, chemicals or drugs, surgery, hormonal change, or other stress events within the previous 30 to 45 days may act as a potential trigger for immune-mediated hemolytic anemia (IMHA)



TREATMENT

HEALTH CARE

- Inpatient during the sudden (acute) hemolytic crisis, during which the body is destroying red-blood cells; outpatient when the packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood) has stabilized, ongoing breakdown of red-blood cells (hemolysis) has been controlled, and clinical signs of low red-blood cell count (anemia) have resolved
- Inpatient if animal has complications such as development of a blood-clotting disorder (disseminated intravascular coagulopathy or DIC); blood clots to the lungs (known as “pulmonary thromboembolism”); low platelet counts (thrombocytopenia); bleeding into the gastrointestinal tract; and heart failure and/or the need for multiple transfusions
- Long-term (chronic), low-grade breakdown of red-blood cells outside of the blood vessels (known as “extravascular hemolysis”) can be treated on an outpatient basis, if the patient is not exhibiting clinical signs secondary to the low red-blood cell count (anemia)
- Fluid therapy to maintain vascular volume and correct dehydration; use caution with administering fluids to patients with long-term (chronic) low red-blood cell counts (anemia) because volume overload (that is, too much fluid) is a concern
- Address underlying cause (such as infection or medications), if secondary immune-mediated hemolytic anemia (IMHA) is diagnosed

ACTIVITY

- Cage rest until stable

SURGERY

- Surgical removal of the spleen (known as “splenectomy”) can be considered if medical management fails to control the disease after 4 to 6 weeks of treatment



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Cross-matched, packed red-blood cells for transfusion or oxyhemoglobin in cases with severely low red-blood cell counts (severe anemia)
- Supportive treatment for animals with blood-clotting disorder (disseminated intravascular coagulopathy or DIC), such as heparin or ultra-low dose aspirin along with azathioprine and steroids significantly improve survival
- Steroids—prednisone, initially at a high dose and then gradually tapered to the lowest effective dose; follow the dosage prescribed by your pet's veterinarian carefully; dexamethasone can be used instead of prednisone; follow similar tapering schedule
- Chemotherapeutic drugs, if clumping together of red-blood cells due to the presence of antibodies (known as “autoagglutination”) or very sudden breakdown of red-blood cells (known as “peracute hemolysis”) exists or if the response to prednisone is poor after 14 to 21 days; drugs include azathioprine, cyclophosphamide, or chlorambucil (for cats)
- Studies have shown no increased effectiveness with combination therapy of cyclophosphamide and prednisone versus using prednisone alone
- Lefluomide may be useful in cases that do not respond to medical treatment
- Cyclosporine—decreases the immune response
- Danazol—for dogs; not recommended in cats as drug may cause liver damage

- Human γ -globulin



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor heart rate, breathing rate, and body temperature frequently during hospitalization
- Monitor for adverse reactions to treatment (such as transfusion reactions and overhydration [that is, too much fluid])
- If blood clots to the lungs (pulmonary thromboembolism) are suspected, frequently monitor chest X-rays and arterial blood gases (measurements of oxygen and carbon dioxide levels in arterial blood)
- During the first month of treatment, check the packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood) weekly until stable and then every 2 weeks for 2 months; if still stable, recheck PCV monthly for 6 months and then 2 to 4 times per year; rechecks may need to be more frequent if patient is on long-term medication
- A complete blood count (CBC) should be rechecked at least monthly during treatment, especially if chemotherapeutic drugs are used; if the neutrophil count falls to below 3,000 cells/mL of blood, discontinue chemotherapeutic drugs until the count recovers and then start drugs at a lower dosage; “neutrophils” are a type of white-blood cell that fight infection
- Blood tests (such as reticulocyte count and Coombs’ test) can be monitored if the packed cell volume (“PCV,” a means of measuring the percentage volume of red blood cells as compared to the fluid volume of blood) is not rising as expected

POSSIBLE COMPLICATIONS

- Blood clots in the lungs and other organs (known as “pulmonary and multiorgan thromboembolism”) have been identified in up to 80% of all cases at necropsy
- Blockage of the portal vein by a blood clot; the portal vein is the vein carrying blood from the digestive organs to the liver (condition known as “portal vein thrombosis”)
- Blood-clotting disorder (disseminated intravascular coagulopathy or DIC); has been diagnosed in 32% of dogs with immune-mediated hemolytic anemia (IMHA)
- Irregular heart beats (known as “cardiac arrhythmias”); death of tissues in the liver (known as “centrilobular hepatic necrosis”); and death of kidney tubules (known as “renal tubular necrosis”) secondary to low levels of oxygen in the blood and/or tissues (known as “hypoxia”)
- Secondary infection and inflammation/infection of the heart (known as “endocarditis”)
- Death

EXPECTED COURSE AND PROGNOSIS

- Immune-mediated hemolytic anemia (IMHA) and its complications (such as a blood-clotting disorder [disseminated intravascular coagulopathy or DIC] and blood clots to the lungs [pulmonary thromboembolism]) can be fatal
- Very sudden (known as “peracute”) disease usually caused by clumping together of red-blood cells due to the presence of antibodies (autoagglutination) or breakdown of red-blood cells within blood vessels (known as “intravascular hemolysis”)
- Sudden (acute) disease usually caused by breakdown of red-blood cells within blood vessels (intravascular hemolysis) or outside of blood vessels (extravascular hemolysis)
- Long-term (chronic) disease usually caused by breakdown of red-blood cells outside of blood vessels (extravascular hemolysis) or cold-reacting antibodies (as identified in the laboratory)
- Increased levels of bilirubin (a bile pigment formed from hemoglobin) in the blood (known as “hyperbilirubinemia”) measured at greater than 5 mg/dL on blood work; clumping together of red-blood cells due to the presence of antibodies (autoagglutination); breakdown of red-blood cells within blood vessels (intravascular hemolysis); severely low platelet counts (thrombocytopenia); low levels of albumin, a protein, in the blood (known as “hypoalbuminemia”) are associated with a poor prognosis
- Overall mortality 33.3% in dogs; reported as 23.5% in cats
- Clumping together of red-blood cells due to the presence of antibodies (autoagglutination) is associated with up to 50% mortality
- Very sudden (peracute) breakdown of red-blood cells (hemolysis) is associated with up to 80% mortality
- Warm-reacting immune-mediated hemolytic anemia (IMHA) has a guarded prognosis; of patients who survive hospitalization (up to 71%), long-term prognosis is relatively good
- Cold-reacting immune-mediated hemolytic anemia (IMHA) is more resistant to drugs to decrease the immune response (known as “immunosuppressive drugs”) than warm-reacting IMHA
- Response to treatment may take weeks to months; immune-mediated hemolytic anemia (IMHA) in which the bone marrow does not respond adequately to produce more red-blood cells (known as “nonregenerative anemia”) may have a more gradual onset than typical IMHA and may be slower to respond to treatment
- Breakdown of red-blood cells (hemolysis) may recur, despite previous or current treatment



KEY POINTS

- Immune-mediated hemolytic anemia (IMHA) and its complications (such as a blood-clotting disorder [disseminated intravascular coagulopathy or DIC] and blood clots to the lungs [pulmonary thromboembolism]) can be fatal

- Life-long treatment may be needed, and the disease may recur
- Side effects of treatment may be severe

ANISOCORIA

(UNEQUAL PUPIL SIZE)



BASICS

OVERVIEW

- The pupil is the circular or elliptical opening in the center of the iris of the eye; light passes through the pupil to reach the back part of the eye (known as the “retina”); the iris is the colored or pigmented part of the eye—it can be brown, blue, green, or a mixture of colors
- The pupil constricts or enlarges (dilates) based on the amount of light entering the eye; the pupil constricts with bright light
- “Anisocoria” is an inequality of pupil size in the animal (in other words, one pupil is larger than the other)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Unequal pupils
- May have other signs, based on the underlying cause

CAUSES

Nervous System Disorders

- Disease affecting nerves to eye (the optic nerve, optic tract, and oculomotor nerve) or part of the brain (known as the “cerebellum”)

Ocular (Eye) Disorders

- Inflammation of the front part of the eye, including the iris (known as “anterior uveitis”)
- Disease of the eye, in which the pressure within the eye is increased (known as “glaucoma”)
- Decrease in iris tissue (known as “iris atrophy”) or poorly developed iris (known as “iris hypoplasia”)
- Scar tissue between the iris and the lens of the eye (known as “posterior synechia”); the lens is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye (retina)
- Medications (such as atropine applied to only one eye, causing that pupil to enlarge or dilate)
- Cancer
- Spastic pupil syndrome—condition in which pupils alternatively will be unequal in size and then normal in size; may be associated with feline leukemia virus (FeLV) infection in cats



TREATMENT

HEALTH CARE

- Depends on underlying disease



MEDICATIONS

- Depends on underlying disease



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on underlying disease

PREVENTIONS AND AVOIDANCE

- Depends on underlying disease

POSSIBLE COMPLICATIONS

- Depends on underlying disease

EXPECTED COURSE AND PROGNOSIS

- Depends on underlying disease



KEY POINTS

- The pupil is the circular or elliptical opening in the center of the iris of the eye
- “Anisocoria” is an inequality of pupil size in the animal (in other words, one pupil is larger than the other)

LACK OF APPETITE (ANOREXIA)



BASICS

OVERVIEW

- Lack or loss of appetite for food; may be partial or complete lack of appetite; anorexia results in decreased food intake, which then leads to weight loss
- Hunger is aroused by the body's need for food
- "Pseudoanorexia" is difficulty in taking hold of food or swallowing, which results in decreased food intake rather than an actual loss of appetite; "pseudo-" refers to "false," so the term "pseudoanorexia" is "false lack of appetite"

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Refusal to eat; poor appetite is associated strongly with illness
- Patients with disorders causing dysfunction or pain of the face, neck, throat (oropharynx), and esophagus (the tube from the throat to the stomach) may display interest in food, but cannot eat—these patients are referred to as being "pseudoanorectic"
- Animals lacking sense of smell (known as "anosmia") often show no sniffing behavior
- Weight loss
- Clinical signs vary depending on the underlying cause; they may include fever; pale gums and other tissues of the body; yellowish discoloration to the gums and moist tissues of the body (known as "jaundice" or "icterus"); pain; changes in organ size; changes in the eyes; abdominal swelling or enlargement; difficulty breathing (known as "dyspnea"); muffled heart and lung sounds; abnormal lung sounds; heart murmurs, and the presence of tumors
- "Pseudoanorectic" patients commonly display weight loss; bad breath (known as "halitosis"); excessive drooling; difficulty in taking hold of food and chewing food; and painful swallowing

CAUSES

Lack of Appetite (Anorexia)

- Almost any generalized (systemic) disease process can cause lack of appetite (anorexia)
- Psychological lack of appetite—food that is not tasty; food that the animal dislikes or avoids (known as "food aversion"); stress, alterations in routine and environment
- Abnormalities in the pH of the blood and body tissues (known as "acid-base disorders")
- Heart failure
- Poisons and medications
- Pain
- Hormonal (endocrine) disease and disease caused by accumulation of chemicals or compounds normally produced by the body (known as "metabolic disease")
- Cancer
- Infectious disease (such as viral or bacterial disease)
- Diseases caused by abnormal immune response (known as "immune-mediated disease")
- Diseases of the airways and lungs (known as "respiratory disease") or of the stomach and intestines (known as "gastrointestinal disease")
- Diseases of the muscles, bones, and nerves
- Lack of appetite related to aging
- Miscellaneous causes, such as motion sickness, high environmental temperature

False Lack of Appetite (Pseudoanorexia)

- Any disease causing pain or difficulty in taking hold of food and chewing food and swallowing
- Inflammation of the mouth (known as "stomatitis"); inflammation of the tongue (known as "glossitis"); inflammation of the gums (known as "gingivitis"); inflammation of the throat (known as "pharyngitis"); and inflammation of the esophagus (the tube from the throat to the stomach; condition known as "esophagitis")—these can be caused by physical agents, caustic materials, bacterial or viral infections, foreign bodies, immune-mediated diseases, excess levels of urea and other nitrogenous waste products in the blood (known as "uremia" or "azotemia")
- Disorders involving the tissues behind the throat (known as "retropharyngeal disorders"), such as enlarged lymph nodes (known as "lymphadenopathy"); abscess; localized accumulation or mass of blood (known as a "hematoma"); or cyst containing saliva (known as a "sialocele")
- Dental disease or inflammation/infection of the gums and supporting tissues of the teeth (known as "periodontal disease")
- Abscess located behind the eyeball (known as "retrobulbar abscess")
- Cancer involving the mouth, tongue, throat, or esophagus (the tube from the throat to the stomach)
- Nervous system disorders (such as rabies and central nervous system lesions)

- Disorders of the muscles and/or bones of the head (examples include masticatory myositis, temporomandibular joint disease, fractures, craniomandibular osteopathy, myasthenia gravis, botulism, and cricopharyngeal achalasia)
- Salivary gland cancer or inflammation



TREATMENT

HEALTH CARE

- The mainstay of treatment is aimed at identifying and correcting the underlying disease
- Symptomatic therapy includes attention to fluid and electrolyte needs, reduction in environmental stressors, and modification of the diet to improve tastiness (palatability)
- Tastiness (palatability) can be improved by adding flavored toppings (such as chicken and beef broth), seasoning with condiments (such as garlic powder), increasing the fat or protein content of the food, and heating the food to body temperature
- As a general rule, dogs and cats with debilitating disease should not go without food for longer than 3 to 5 days before starting some type of supplemental feeding (such as forced feeding, tube feeding or intravenous feeding)

DIET

- Tasty (palatable) diet
- May try different consistencies of food—gruel; hard food; soft food; evaluate what is best accepted

SURGERY

- Surgical placement of a feeding tube may be necessary



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Diazepam and oxazepam are short-acting appetite stimulants (with sedative properties); used in cats
- Cyproheptadine, an antihistamine, has been used as an appetite stimulant with mixed success (give 10 to 20 minutes prior to feeding)
- Medications to relieve pain (known as “analgesics”) may promote appetite in painful conditions
- Metoclopramide, ranitidine, or erythromycin may be useful, if lack of appetite (anorexia) is associated with abnormalities in the muscles or function of the stomach or intestines
- Medications to control vomiting or nausea (known as “antiemetics”) are useful to decrease nausea-associated lack of appetite (anorexia); examples include prochlorperazine or metoclopramide



FOLLOW-UP CARE

PATIENT MONITORING

- Body weight, body condition score (BCS) assessment (estimate of weight status [under or overweight] as compared to normal weight), and hydration determination
- Return of appetite

PREVENTIONS AND AVOIDANCE

- Feed highly tasty (palatable) diet

POSSIBLE COMPLICATIONS

- Dehydration, malnutrition, and extreme weight loss with muscle wasting (known as “cachexia”) are most likely; these complications can increase the severity of the underlying disease
- A loss of more than 25% to 30% of body protein negatively affects the immune system and muscle strength, and death results from infection and/or lung failure
- Disease in which fats and lipids (compounds that contain fats or oils) accumulate in the liver (condition known as “feline hepatic lipidosis”) is a possible complication of lack of appetite (anorexia) in obese cats
- Breakdown of the intestinal lining (mucosal) barrier is a concern in debilitated patients; the intestinal mucosal barrier protects the body as it acts to prevent bacteria and toxins from entering the body through the intestinal tract

EXPECTED COURSE AND PROGNOSIS

- Varies with underlying cause



KEY POINTS

- Lack or loss of appetite for food; may be partial or complete lack of appetite; anorexia results in decreased food intake, which then leads to weight loss
- Hunger is aroused by the body's need for food
- "Pseudoanorexia" is difficulty in taking hold of food or swallowing, which results in decreased food intake rather than an actual loss of appetite
- The mainstay of treatment is aimed at identifying and correcting the underlying disease

ANTEBRACHIAL GROWTH DEFORMITIES

(ABNORMAL DEVELOPMENT OF THE FORELEG IN GROWING ANIMALS)



BASICS

OVERVIEW

- Abnormally shaped forelimbs and/or displacement of the elbow or carpus (joint between front paw and foreleg) out of normal alignment that result from abnormal development of the bones (radius or ulna) in the foreleg in the growing animal

GENETICS

- Skye terriers—reported as a recessive inheritable trait
- Dogs, such as basset hounds and dachshunds, with “normal” short, bowed legs are known as “chondrodysplastic breeds;” these breeds may be more likely to have improper alignment of the bones (malalignment) in the elbow than other breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Skye terriers—recessive inheritable form
- Chondrodysplastic and toy-breed dogs (especially basset hounds, dachshunds, Lhasa apsos, Pekingese, Jack Russell terriers)—may be more likely to have improper alignment of the bones (malalignment) in the elbow than other breeds
- Giant-breed dogs (such as Great Danes, wolfhounds)—abnormal development may be induced by rapid growth, owing to excessive or unbalanced nutrition, or by bone disorders in young, growing dogs, such as osteochondritis dissecans (OCD) or hypertrophic osteodystrophy (HOD)

Mean Age and Range

- Traumatic causes can lead to foreleg (antebrachial) growth deformities anytime during the active growth phase of the bones
- Abnormal elbow joint actually occurs during growth; however, it may not be recognized until secondary arthritic changes become severe, occasionally at several years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

The foreleg is composed of two long bones—the radius and the ulna. The long bones in the body grow in length at specific areas known as “growth plates.” These areas usually continue to produce bone until the bones are fully developed, at which time, no further growth is needed. The growth plates then “close” and become part of the bone. If something happens to the growth plate before the bone is fully developed, it may close prematurely. If only one of the two bones in the foreleg is affected, the other bone continues to grow, causing the foreleg to become deformed. If only one side of a growth plate closes prematurely, the bone will continue to grow on the opposite side, leading to curving of the bone. Signs and physical examination findings are determined by which bone is affected initially and by the location of the premature closure of the growth plate.

- Longer-limbed dogs—foreleg deformities generally more common
- Shorter-limbed dogs—tend to develop more severe joint malalignments
- Age at the time of premature closure of the bone growth plates affects the relative degree of foreleg deformity and joint malalignment
- Trauma to the forelimb in a growing animal—progressive limb deformity or lameness 2–4 weeks after injury; owner may not be aware of causative event
- Developmental elbow malalignments—slowly progressive onset of lameness in one or both forelimbs; most apparent after exercise

Physical Examination Findings

1. Premature Closure of the Growth Plate of the Radius

- Affected limb—significantly shorter than the normal opposite forelimb
- Severity of lameness—depends on degree of joint abnormality
- Complete symmetrical closure of lower growth plate of the radius—may note straight limb with a widened space in the joint between the radius and the carpus (radial carpal joint); may note caudal bow to radius and ulna
- Asymmetrical closure of medial lower growth plate of the radius—foreleg deformity in which the foreleg is twisted or bent inward toward the center of the body (varus angular deformity); occasionally inward rotation
- Closure of lateral lower growth plate of the radius—foreleg deformity in which the foreleg is twisted or bent outward away from the center of the body (valgus angular deformity); external rotation
- Closure of upper growth plate of the radius with continued growth of the ulna—deformity of the elbow joint with increased spaces between the bones of the elbow

2. Premature Closure of the Growth Plate of the Lower Ulna (Distal Ulna)

- Three deformities of the lower (distal) radius may result, leading to the foreleg being twisted or bent to the side (valgus deformity), bowing toward the front of the leg (cranial bowing or curvus deformity), and external rotation (supination)
- Relative shortening of limb length compared to the opposite normally growing limb
- Partial dislocation (caudolateral subluxation) of the joint between the radius and the carpus (radial carpal joint) and abnormal motion of the elbow joint due to improper alignment of the bones (malalignment) may occur; causes lameness and painful restriction of joint

movement

CAUSES

- Trauma
- Developmental basis
- Nutritional basis

RISK FACTORS

- Forelimb trauma
- Excessive dietary supplementation



TREATMENT

HEALTH CARE

- Genetic predisposition—do not breed animals with abnormal growth development of the foreleg
- Traumatic growth plate damage—not seen at time of injury; revealed 2–4 weeks later
- Surgical treatment is recommended as soon as possible following diagnosis

ACTIVITY

- Exercise restriction—reduces joint damage; slows arthritic progression

DIET

- Decrease nutritional supplementation in giant-breed dogs—slows rapid growth; may reduce likelihood of abnormal development of foreleg
- Avoid excess weight—helps control arthritic pain resulting from joint malalignment and overuse

SURGERY

- Premature growth plate closure of the lower ulna in a patient less than 5–6 months of age (with significant amount of radial growth potential remaining)—treated with a surgical procedure in which a section of the ulna is removed (segmental ulnar ostectomy) to allow the ulna to lengthen as the radius continues to grow in length, valgus deformities of 25° or less may spontaneously correct and may not require additional surgery; many patients as well as those with more severe deformities often require a second surgical correction after they have matured
- Premature growth plate closure of the radius or ulna seen in a mature patient (that is, one with limited or no growth potential) requires surgical deformity correction, joint realignment, or both
- Deformity correction—may be accomplished with a variety of surgical bone-cutting techniques; the bone then may be stabilized with fixation devices; must correct both rotational and angular deformities; the surgery is performed at the point of greatest curvature
- Joint malalignment (particularly elbow)—must correct to minimize arthritic development (primary cause of lameness); obtain optimal joint alignment using a surgical bone cutting technique or by shortening longer of the two bones
- Significant limb length discrepancies—surgical bone-cutting technique of the shortened bone, which is then progressively and slowly lengthened (distracted) using an external fixator system (known as “distraction osteogenesis”) to create new bone length



MEDICATIONS

- Anti-inflammatory drugs—symptomatic treatment of arthritis
- Nutraceuticals (such as glucosamine) may help minimize cartilage damage and arthritis development; may be anti-inflammatory and may relieve pain



FOLLOW-UP CARE

PATIENT MONITORING

- Postoperative—depends on surgical treatment
- Periodic checkups—evaluate arthritic status and anti-inflammatory therapy

PREVENTIONS AND AVOIDANCE

- Selective breeding of susceptible breeds
- Avoid dietary over supplementation in rapidly growing giant-breed dogs

POSSIBLE COMPLICATIONS

- Routinely seen with various surgical bone-cutting and fixation techniques (such as infection, lack of healing of the bone [nonunion of osteotomy], metal pin [fixator] tract inflammation)

EXPECTED COURSE AND PROGNOSIS

- Generally, best results seen with early diagnosis and surgical treatment—minimizes arthritis
- Premature ulnar closure—tends to be easier to manage; yields better results
- Limb lengthening by distraction osteogenesis—requires extensive postoperative management by the veterinarian and owner; high rate of complications



KEY POINTS

- Antebrachial growth deformities are reported to be inherited in Skye terriers and may be inherited in the chondrodysplastic breeds of dogs
- Damage to the growth plate is not apparent at time of forelimb trauma and diagnosis is often made at 2–4-weeks following an injury
- Joint malalignment and resultant arthritis are primary causes of lameness
- Early surgical treatment generally leads to a better prognosis

ANTICOAGULANT RODENTICIDE POISONING



BASICS

OVERVIEW

- An “anticoagulant” is something that prevents blood from clotting; a “rodenticide” is a product that kills rodents (such as mice and rats)
- Blood-clotting disorder (known as a “coagulopathy”) caused by reduced vitamin K₁-dependent clotting factors in the circulation after exposure to anticoagulant rodenticides
- “Clotting factors” are components in the blood involved in the clotting process—the clotting factors are identified by Roman numerals, I through XIII

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Difficulty breathing (known as “dyspnea”)
- Bleeding
- Localized mass of blood in a tissue or organ (known as a “hematoma”)—often along the lower areas of the body (known as the “ventrum”) and at sites where intravenous catheters were placed or blood was drawn (known as “venipuncture sites”); may have multiple hematomas
- Muffled heart or lung sounds
- Pale gums and moist tissues of the body (known as “mucous membranes”)
- Sluggishness (lethargy)
- Depression

CAUSES

- Exposure to anticoagulant rodenticide products
- First-generation coumarin anticoagulants (such as warfarin, pindone)—largely replaced by more potent second-generation anticoagulants
- Second-generation anticoagulants (such as brodifacoum, bromadiolone, diphacinone, and chlorophacinone)—generally more toxic and persist much longer in the animal’s body than first-generation agents
- Difenthiolone (D-Cease™ Mouse and Rat Bait Pellets)—highly toxic to mice and rats; less toxic to dogs than are brodifacoum, bromadiolone, chlorophacinone, and warfarin

RISK FACTORS

- Use of anticoagulant rodenticides
- Anticoagulant rodenticide poisoning may be slightly more likely in the spring and fall, when rodenticide products are used
- Small doses over several days more dangerous than a single large dose; either type of exposure may cause bleeding problems
- Secondary poisoning by consumption of poisoned rodents—unlikely



TREATMENT

HEALTH CARE

- Inpatient—sudden (acute) crisis
- Outpatient—consider once the blood-clotting disorder (coagulopathy) is stabilized
- Fresh whole blood or plasma transfusion—may be required if pet is bleeding; provides immediate access to vitamin K-dependent clotting factors; whole blood may be preferred with severely low red-blood cell count (known as “severe anemia”) from sudden (acute) or long-term (chronic) blood loss

ACTIVITY

- Confine patient during the early stages; activity enhances blood loss

DIET

- No recognized effect

SURGERY

- Procedure to tap the chest (known as “thoracocentesis”)—may be important for removing free blood in the space between the chest wall and lungs (known as the “pleural space”), which causes difficulty breathing (dyspnea) and breathing failure
- Must correct blood-clotting disorder (coagulopathy) before surgery



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Vitamin K₁—administered by mouth, as directed by your pet’s veterinarian; length of treatment depends on the specific anticoagulant rodenticide product to which the animal was exposed; feeding of a small amount of fat, such as canned dog food, helps absorption of vitamin K₁



FOLLOW-UP CARE

PATIENT MONITORING

- Blood tests (activated clotting time [ACT] and prothrombin time [PT]) to evaluate clotting status—assess effectiveness of therapy; monitoring continued 3 to 5 days after discontinuation of vitamin K₁ treatment

PREVENTIONS AND AVOIDANCE

- Do not allow animals to have access to anticoagulant rodenticides

POSSIBLE COMPLICATIONS

- Secondary bacterial pneumonia after bleeding into the lungs
- Death

EXPECTED COURSE AND PROGNOSIS

- Patient survives the first 48 hours of sudden (acute) blood-clotting disorder (coagulopathy)—prognosis improves



KEY POINTS

- Anticoagulant rodenticide poisoning is a common problem—many rodent baits are sold over the counter and widely used in homes
- Re-exposure of the animal to anticoagulant rodenticides could be a serious problem
- Do not allow animals to have access to anticoagulant rodenticides

AORTIC STENOSIS



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- “Stenosis” is the medical term for narrowing
- “Aortic stenosis” is a narrowing at some point in the area through which blood flows out of the left ventricle and through the aortic valve and into the aorta (known as the “left ventricular outflow tract”), most commonly seen as a congenital (present at birth) or perinatal (occurring at or around birth) disease
- Defect can be at the valve itself (known as “valvular aortic stenosis”), below the valve (known as “subvalvular aortic stenosis” or “subaortic stenosis” [most common in dogs]), or above the valve, just inside the aorta (known as “supravalvular aortic stenosis” [most common in cats])
- As a congenital (present at birth) structural abnormality in dogs, the obstruction usually is caused by fibrous tissue just below the aortic valve, and the disease is referred to as subaortic stenosis (SAS)

GENETICS

- Inherited trait in Newfoundlands—involves multiple genes and exhibits pseudodominance; a major dominant gene with modifiers may be involved
- Genetic history of affected litters from the same male dog (known as the “sire”) or female dog (known as the “bitch”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Subaortic stenosis is most common in the Newfoundland, German shepherd dog, golden retriever, rottweiler, and boxer; the Samoyed, English bulldog, and Great Dane also are at higher risk of having subaortic stenosis than are other breeds
- Bull terriers are more likely than other breeds to have valvular aortic stenosis, typically with coexistent abnormal development of the mitral valve (known as “mitral valve dysplasia”)

Mean Age and Range

- Subaortic stenosis develops over the first weeks to months of life; onset of clinical signs can occur at any age, depending on the severity of blockage or obstruction to blood flow from the left ventricle into the aorta (the main artery of the body)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Related to the severity of the narrowing or blockage of blood flow; range from no signs to congestive heart failure, fainting (known as “syncope,”) and sudden death; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Heart murmur; murmur may not be present in puppies less than 2 months old, becoming more prominent during the first 6 months of age
- May be able to feel vibrations caused by abnormal blood flow (known as “thrills”) when placing hand against the chest wall
- Difficulty breathing (known as “dyspnea”), rapid breathing (known as “tachypnea”), and short, rough snapping sounds (known as “crackles”) detected when listening to the lungs with a stethoscope (known as “auscultation”) with the onset of left-sided congestive heart failure
- Femoral pulses typically weakened in animals with disease severe enough to affect circulation
- Irregular heart beats (known as “arrhythmias”)

CAUSES

- Congenital (present at birth) disease
- Secondary to bacterial inflammation/infection of the lining of the heart (known as “bacterial endocarditis”), involving the aortic valve, in some dogs
- In cats with hypertrophic cardiomyopathy (a disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle), functional or dynamic narrowing (stenosis) is common, but significance is unknown; a “dynamic” process in one in which the lumen of the ventricle changes with the movements of the heart (relaxation and contraction)
- “Dynamic” subaortic stenosis reported in dogs in which muscular enlargement or thickening (known as “hypertrophy”) can contribute to narrowing of the ventricular/aortic outflow tract

RISK FACTORS

- Familial (runs in certain families or lines of animals) history of subaortic stenosis
- Inflammation/infection of the lining of the heart and aortic valve (known as “aortic endocarditis”) is more likely in animals with decreased ability to produce a normal immune response (known as “immunosuppression”); generalized (systemic) infection; presence of

bacteria in the blood (known as “bacteremia”); and abnormal blood flow within the heart, including congenital (present at birth) subaortic stenosis



TREATMENT

HEALTH CARE

- Management recommendations for small animals are controversial and vary among experts
- Inpatient management is appropriate for complications including irregular heart beats (arrhythmias), episodes of fainting (syncope), and congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

ACTIVITY

- Restricted in animals with more than mild disease
- Fainting (syncope), collapse, or sudden death may be brought on by exertion in animals with severe disease

DIET

- Restricted sodium in animals in or likely to develop congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

SURGERY

- Definitive treatment requires open-heart surgery with cardiopulmonary bypass (using a heart-lung machine) to repair or replace the valve; unfortunately, the risk-to-benefit ratio for dogs with subaortic stenosis currently does not support recommending the surgery—dogs still may die suddenly after the procedure
- Balloon dilation (procedure in which an instrument with an expandable balloon is inserted into the aorta and the balloon is expanded to open the narrowing) of the outflow tract during heart catheterization may result in improvement of clinical signs in some dogs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medical management is, at best, designed to treat the signs—it does not cure the condition
- Beta-blockers have been advocated for dogs with subaortic stenosis with a history of fainting (syncope) or collapse and for irregular heart beats (arrhythmias) that occur following exercise; potential benefits include limiting the oxygen needs of the heart muscle, protecting against irregular heart beats (arrhythmias), and slowing of the heart rate; an example of a beta blocker is atenolol; metoprolol tartrate and carvedilol are alternative beta-blockers
- Specific treatment for irregular heart beats (such as ventricular arrhythmias and atrial fibrillation) or left-sided congestive heart failure also may be required; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Affected animals are at risk of developing bacterial inflammation/infection of the lining of the heart (bacterial endocarditis); treatment of infections with antibiotics is recommended; antibiotics should be administered for animals having dental procedures or surgery of the genitourinary tract (that is, surgery involving the reproductive tract and/or urinary tract)
- Treatment of dynamic narrowing (stenosis) in cats with hypertrophic cardiomyopathy (a disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle) is controversial; a “dynamic” process in one in which the lumen of the ventricle changes with the movements of the heart (relaxation and contraction)
- Diltiazem (a calcium-channel blocker heart medication) may have benefits in treating aortic stenosis



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor by electrocardiogram (“ECG,” a recording of the electrical activity of the heart), chest X-rays, and echocardiography (use of ultrasound to evaluate the heart and major blood vessels)
- Treatment of complications (such as congestive heart failure and irregular heart beats [arrhythmias]) necessitates careful monitoring to detect kidney and electrolyte disturbances and side effects of drugs

POSSIBLE COMPLICATIONS

- Congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body's needs)
- Irregular heart beats (arrhythmias); sudden lack of blood supply to the heart muscle that leads to death of tissues (known as "myocardial infarction"); backward flow of blood through the aortic valve (known as "aortic regurgitation"); backward flow of blood through the mitral valve (known as "mitral regurgitation"); bacterial inflammation/infection of the lining of the heart (bacterial endocarditis); sudden death

EXPECTED COURSE AND PROGNOSIS

- Mildly affected dogs may live a normal life span without treatment
- Severe disease typically limits longevity due to congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body's needs) or sudden death
- Congestive heart failure, collapse, or fainting (syncope) suggests severe disease and an ominous prognosis



KEY POINTS

- Affected animals should be neutered or otherwise not be permitted to breed
- Evaluate closely related dogs for evidence of clinical disease
- Mildly affected dogs may live a normal life span without treatment
- Monitor for potential complications in severely affected animals
- Severe disease typically limits longevity due to congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body's needs) or sudden death
- Affected animals have increased risk for bacterial inflammation/infection of the lining of the heart (bacterial endocarditis) and anesthetic complications

AORTIC THROMBOEMBOLISM

(BLOOD CLOTS IN THE AORTA)



BASICS

OVERVIEW

- “Aortic” refers to the aorta, the main artery of the body; “thromboembolism” is blockage of blood flow secondary to the presence of a blood clot in an artery
- “Aortic thromboembolism” results from a blood clot (known as a “thrombus”) that is dislodged within the aorta, causing severely reduced blood flow to the tissues receiving blood from that particular part of the aorta, leading to decreased oxygen in the tissues (reduced blood flow leading to decreased oxygen in the tissues is known as “ischemia”)
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles

GENETICS

- Although aortic thromboembolism is not commonly thought of as an inherited disease, a commonly associated disease, “hypertrophic cardiomyopathy” (a disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle) likely is inherited
- A family of domestic shorthair (DSH) cats with hypertrophic cardiomyopathy recently were reported to have died from aortic thromboembolism

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats, rarely dogs

Breed Predispositions

- Mixed-breed cats most commonly are affected
- Abyssinian, Birman and ragdoll purebred cats were reported in one study to have a higher number of cases than would be expected normally

Mean Age and Range

- Mean age is approximately 8 years
- Age range is 1 to 20 years

Predominant Sex

- Males are affected twice as frequently as females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden (acute) onset paralysis and pain are the most common clinical signs
- Weakness or paralysis of the rear legs; occasionally weakness of a front leg
- Lameness or gait abnormality
- Rapid breathing (known as “tachypnea”) or breathing distress is common
- Difficulty breathing (known as “dyspnea”)
- Vocalization and anxiety
- Vomiting
- Absent or diminished femoral pulses
- Bluish or pale nail beds and foot pads
- Low body temperature (known as “hypothermia”) is common
- Heart murmur, irregular heart beats (known as “arrhythmias”) or gallop sound (sequence of three heart sounds heard when listening to the heart with a stethoscope; heart beat sounds like a galloping horse instead of normal “lub-dub”)

CAUSES

- Heart muscle disease (known as “cardiomyopathy”)
- Increased levels of thyroid hormone in the body (known as “hyperthyroidism”)
- Cancer
- Generalized bacterial infection (known as “sepsis”) in dogs
- Increased levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”) in dogs
- Disease in which proteins are lost from the body through the kidneys (known as “protein-losing nephropathy”) in dogs

RISK FACTORS

- Markedly enlarged left atrium
- Blood clot within the chambers of the heart



TREATMENT

HEALTH CARE

- Initially, treat cats as inpatients, because many have coexistent congestive heart failure as well as having considerable pain and distress; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Fluid therapy administered cautiously, as many cats are in congestive heart failure
- Supplemental oxygen therapy or a medical procedure to tap the chest (known as “thoracocentesis”) may be beneficial, if pet is in congestive heart failure
- Initially, the affected legs should be handled minimally; however, as blood flow returns, physical therapy (passive extension and flexion of the legs) may speed full recovery
- Initially, these cats may have difficulty posturing to urinate and may need to have their bladders expressed to prevent over-distention or development of skin lesions due to contact with urine, when the hair and skin remain damp (known as “urine scald”)

ACTIVITY

- Activity should be restricted
- Keep the cat quiet and stress-free

DIET

- Initially, most cats have lack of appetite (known as “anorexia”)
- Tempt these cats with any type of food
- It is important to keep these cats eating to avoid hepatic lipidosis (a disease in which fats and lipids [compounds that contain fats or oils] accumulate in the liver)

SURGERY

- Surgical removal of the blood clot typically is not recommended as these are high-risk patients because of severe heart disease



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medications to break up existing clots (known as “thrombolytic drugs,” such as urokinase, streptokinase or tissue plasminogen activator) are used extensively in people and infrequently in cats; these drugs are expensive and carry a significant risk for bleeding complications and thus rarely are used in general veterinary practice
- Heparin is the preferred drug in general practice; it has no effect on the established clot; however, it prevents further clotting
- Aspirin theoretically is beneficial during and after an episode of aortic thromboembolism because of its antiplatelet effects; “platelets” are normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding; if they accumulate in a blood vessel, they may lead to a blood clot (known as “thrombosis”)—aspirin should be administered only under the direction of your pet’s veterinarian
- Clopidogrel, an anti-platelet aggregation drug, has some promise for management and prevention of future aortic thromboembolism
- Buprenorphine is an opiate used to relieve pain (known as “analgesia”) and to sedate the pet; for stronger analgesia, fentanyl or hydromorphone could be used
- Acepromazine may be used cautiously for its sedative effects and to dilate blood vessels (known as “vasodilation”)
- Warfarin, a vitamin-K antagonist, is a medication that decreases blood clotting (known as an “anticoagulant”); it is used most widely in people and has been proposed for prevention of re-embolization in cats surviving an initial episode—long-term management with warfarin can be challenging because of the necessity for frequent monitoring and dose adjustments as well as side effects, such as bleeding
- Low molecular-weight heparin (LMWH) recently has been proposed for the long-term prevention of feline aortic thromboembolism; LMWH has a more predictable relationship between dose and response than warfarin and does not need frequent monitoring or dose adjustments; it also has a lower risk of bleeding complications than warfarin
- Treat the patient’s heart disease; medications determined by type and severity of heart disease



FOLLOW-UP CARE

PATIENT MONITORING

- Electrocardiogram (“ECG,” a recording of the electrical activity of the heart) monitoring is helpful to detect heart problems with

re-establishment of blood flow (known as “re-perfusion injury”) and high levels of potassium in the blood (known as “hyperkalemia”)

- Monitoring blood work (electrolytes and kidney tests) periodically may be helpful to improve management of the heart disease
- Examine the legs daily to assess clinical response
- Blood-clotting tests (such as “activated partial thromboplastin time [APTT], prothrombin time [PT]”) should be performed; decision upon which clotting test is performed and frequency of testing is determined by medication (such as heparin or warfarin) that pet is receiving

PREVENTIONS AND AVOIDANCE

- Prevention of future blood clots in the aorta with long-term (chronic) administration of aspirin, clopidogrel, warfarin, or LMWH is recommended strongly because of the high rate of blood-clot formation

POSSIBLE COMPLICATIONS

- Bleeding with medications to prevent blood clotting (anticoagulant therapy)
- Permanent nervous system deficits or muscular abnormalities in the hind legs
- Recurrent congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Sudden death

EXPECTED COURSE AND PROGNOSIS

- Expected course is days to weeks for full recovery of function to the legs
- Prognosis in general is poor—in two studies, approximately 60% of cats were euthanized or died during the initial episode of aortic thromboembolism
- Long-term prognosis varies between 2 months to several years; however, the average is approximately a few months with treatment
- Predictors of poorer prognosis include low body temperature (hypothermia; less than 99° F), and congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Predictors of better prognosis include normal body temperature (known as “normothermia”), only a single leg affected and presence of ability to move the leg on initial examination
- Recurrence of aortic thromboembolism is common



KEY POINTS

- Be aware of the poor short- and long-term prognosis
- Most cats will develop future blood clots
- Most cats that survive the initial episode will recover complete function of their affected legs; however, if decreased blood flow and lack of oxygen to the tissues (ischemia) was severe and prolonged, sloughing of parts of the lower legs or persistent nervous system deficits may result
- Most cats that survive the initial episode will be on some type of medication to prevent blood clotting (anticoagulant therapy) and may require frequent reevaluations and an indoor lifestyle

ARTHRITIS (OSTEOARTHRITIS)



BASICS

OVERVIEW

- “Arthritis” is the medical term for inflammation of the joints; “osteoarthritis” is a form of joint inflammation (arthritis) characterized by long-term (chronic) deterioration or degeneration of the joint cartilage
- Progressive and permanent deterioration of joint cartilage
- Also known as “degenerative joint disease” or “DJD”

GENETICS

- Primary degenerative joint disease (progressive and permanent deterioration of joint cartilage) is rare—once associated with a colony of beagles
- Dogs—causes of secondary degenerative joint disease (progressive and permanent deterioration of joint cartilage) are varied, including abnormal development of the hip (known as “hip dysplasia”) or elbow (known as “elbow dysplasia”); abnormal development of bone and cartilage, leading to a flap of cartilage within the joint (known as “osteochondritis dissecans” or “OCD”); dislocation of the knee cap (known as a “patellar luxation”); congenital (present at birth) shoulder dislocation (known as a “shoulder luxation”); noninflammatory death of tissue (known as “necrosis”) involving the femoral head (the “ball” of the hip joint) with collapse of the bone (condition known as “Legg-Calvé-Perthes disease”); and cranial cruciate ligament rupture of the stifle or knee
- Cats—causes of secondary degenerative joint disease (progressive and permanent deterioration of joint cartilage) are dislocation of the knee cap (patellar luxation), abnormal development of the hip (hip dysplasia), and any joint disease (known as an “arthropathy”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Secondary degenerative joint disease (progressive and permanent deterioration of joint cartilage) due to congenital (present at birth) disorders (such as hip dysplasia) seen in immature animals; some present with DJD signs when older (such as cases of hip or elbow dysplasia)
- Secondary to trauma—any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Dogs—decreased activity level; unwilling to perform certain tasks; intermittent lameness or stiff gait that slowly progresses
- Possible history of joint trauma; abnormal development of bone and cartilage, leading to a flap of cartilage within the joint (osteochondritis dissecans); or developmental disorders
- Lameness or abnormal gait may become worse with exercise, long periods of lying down or resting, and/or cold weather
- Cats—obvious lameness may not be seen; instead, may have difficulty grooming, jumping onto furniture, or accessing the litter box; may have increased irritability
- Stiff-legged or altered gait (such as “bunny hopping” in hip dysplasia)
- Not using the affected leg(s)
- Decreased range of motion
- Grating detected with joint movement (known as “crepitus”)
- Joint swelling (fluid build-up in the joint [known as “joint effusion”] and/or thickening of the joint capsule)
- Joint pain
- Joint instability
- Obvious joint deformity

CAUSES

- Primary—no known cause (so called “idiopathic osteoarthritis”)
- Secondary—results from an initiating cause, such as abnormal wear on normal cartilage (examples, secondary to joint instability, abnormal joints, trauma to cartilage or supporting soft tissues) or normal wear on abnormal cartilage (example, secondary to defects in the bone and cartilage [known as “osteochondral defects”])

RISK FACTORS

- Working, athletic, and obese dogs place more stress on their joints
- Dogs with disorders that affect collagen or cartilage (such as increased levels of steroids produced by the adrenal glands [known as “hyperadrenocorticism” or “Cushing’s disease”], diabetes mellitus [“sugar diabetes”], inadequate levels of thyroid hormone [known as “hypothyroidism”], excessive looseness of the joints [known as “hyperlaxity”], or prolonged treatment with steroids)



TREATMENT

HEALTH CARE

- Medical treatment—usually tried initially
- Physical therapy—very beneficial
- Maintaining or increasing joint motion—passive range of motion exercises, massage, swimming (as directed by your pet’s veterinarian)
- Pain management—cold and heat therapy, as directed by your pet’s veterinarian
- Muscle tone/strengthening exercises—swimming (aerobic exercise with minimal weight bearing), controlled leash walks up hills or on soft surfaces (such as sand), and dry or water treadmill

ACTIVITY

- Limited to a level that minimizes aggravation of clinical signs

DIET

- Weight reduction for obese patients—decreases stress placed on arthritic joints
- Omega fatty acids may decrease inflammation; use as directed by your pet’s veterinarian

SURGERY

- Surgical options—improve joint geometry or remove bone-on-bone contact areas
- Surgical procedure cutting into or entering a joint (known as an “arthrotomy”)—used to remove aggravating causes (such as bone and/or cartilage fragments or flaps)
- Using a special lighted instrument called an “arthroscope” (general term for procedure is “arthroscopy”) to allow the surgeon to see inside the joint—used to diagnose and remove aggravating causes; flushing the joint may be beneficial
- Reconstructive procedures—used to eliminate joint instability and correct structural or anatomic problems (such as in animals with dislocation of the knee cap [patellar luxation])
- Joint removal—such as removal of the femoral head (the “ball”) of the hip joint for cases of abnormal development of the hip (hip dysplasia; procedure known as “femoral head and neck ostectomy” or “FHO”)
- Joint replacement—total hip replacement is common; total elbow replacement still is experimental
- Joint fusion (known as “arthrodesis”)—in selected long-term (chronic) cases and for joint instability



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs) to decrease pain and inflammation—examples are carprofen, deracoxib, etodolac, meloxicam, tepoxalin
- Medications intended to slow the progression of arthritic changes and protect joint cartilage (known as “chondroprotective drugs”), such as polysulfated glycosaminoglycans, glucosamine, and chondroitin sulfate—may help limit cartilage damage and degeneration; may help alleviate pain and inflammation
- Steroids—decrease inflammation; however, long-term (chronic) use may delay healing and may initiate damage to joint cartilage; examples of steroids are prednisone administered by mouth or triamcinolone administered by injection into the joint (known as an “intra-articular injection”)



FOLLOW-UP CARE

PATIENT MONITORING

- Clinical deterioration—indicates need to change drug selection or dosage; may indicate need for surgical intervention

PREVENTIONS AND AVOIDANCE

- Early identification of conditions that may lead to osteoarthritis and prompt treatment to help reduce progression of secondary conditions

EXPECTED COURSE AND PROGNOSIS

- Slow progression of disease likely
- Medical or surgical treatment usually allows a good quality of life



KEY POINTS

- Medical therapy is designed to control signs of osteoarthritis (known as “palliative treatment”) and not to cure the condition
- Slow progression of disease likely
- Medical or surgical treatment usually allows a good quality of life
- Discuss treatment options, activity level, and diet with your pet’s veterinarian

SEPTIC ARTHRITIS

(INFLAMMATION DUE TO INFECTION OF THE JOINT)



BASICS

OVERVIEW

- Disease-causing bacteria or other microorganisms present within the enclosed space of one or more joints leading to inflammation of the joint (arthritis)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Most common in dogs
- Rare in cats

Breed Predilections

- Medium- to large-breed dogs—most commonly German shepherd dogs, Doberman pinschers, and Labrador retrievers

Mean Age and Range

- Any age; usually between 4 and 7 years of age

Predominant Sex

- Male

SIGNS/OBSERVED CHANGES in the ANIMAL

- Lameness involving a single joint (monoarticular) that is associated with soft-tissue swelling, heat, and pain; rarely lameness involving a few joints (pauciarticular)
- Lameness—sudden (acute) onset is most common, but can present as a long-term (chronic) lameness
- Sluggishness (lethargy)
- Lack of appetite (anorexia)
- Joint pain and swelling—commonly involving the carpus (joint between front paw and foreleg), stifle, hock, shoulder, or elbow joint
- Localized joint heat
- Decreased range of motion
- Fever

CAUSES

- Aerobic bacteria (bacteria that can live and grow in the presence of oxygen)—most common: staphylococci, streptococci, coliforms, and *Pasteurella*
- Anaerobic bacteria (bacteria that can live and grow in the absence of oxygen)—most common: *Propionibacterium*, *Peptostreptococcus*, *Fusobacterium*, and *Bacteroides*
- Spirochete—*Borrelia burgdorferi* (organism that causes Lyme disease)
- *Mycoplasma*
- Fungal agents—*Blastomyces*, *Cryptococcus*, and *Coccidioides*
- *Ehrlichia*
- *Leishmania*

RISK FACTORS

- Predisposing factors for blood-borne infection, such as diabetes mellitus; Addison's disease; diseases or drug therapy that lead to an inability to develop a normal immune response (known as "immunosuppression")
- Previous trauma (such as a dog bite or injury that penetrated the joint) or prior surgery of the joint
- Existing bony arthritis (osteoarthritis) or other joint damage
- Injection into the joint space itself (known as an "intra-articular injection"), particularly if steroid is injected



TREATMENT

HEALTH CARE

- Inpatient—initial stabilization; the veterinarian will initiate systemic antibiotic therapy as soon as joint fluid has been obtained for bacterial culture; the veterinarian may perform joint drainage and flushing (lavage) as soon as possible to minimize injury within the joint
- Identify source if blood-borne spread of the bacteria or microorganisms is suspected
- Outpatient—long-term management

- Alternating heat and cold packing—beneficial in promoting increased blood flow and decreased swelling

ACTIVITY

- Restricted until resolution of signs

SURGERY

- Sudden (acute) disease with minimal changes seen on X-rays—joint drainage and flushing (lavage) via a sterile needle inserted into the joint (arthrocentesis); flushing of the joint via a special instrument or endoscope (arthroscopic lavage) that allows the veterinarian to actually see into the joint; or via a surgical incision into the joint (arthrotomy); an irrigation catheter can be placed in larger joints to allow easier joint flushing
- Chronic disease—may require open surgical incision into the joint (arthrotomy) with removal of abnormal tissue (débridement) of the joint lining (synovium) and copious flushing (lavage); if appropriate, an irrigation catheter may be placed to flush the joint postoperatively
- Flushing (lavage) of the joint—warmed physiologic saline or lactated Ringer’s solution to flush the joint until flushed fluid is clear
- Flushed fluid—monitored daily using a microscope to evaluate existence and character of bacteria and white blood cells (neutrophils)
- Removal of catheters—when flushed fluid has no bacteria and the white blood cells (neutrophils) appear healthy
- Evaluation of the joint via a special instrument or endoscope (arthroscopy) allows for visual assessment of joint cartilage, flushing (lavage) of the joint, and biopsy, and is a less invasive method of thorough joint lavage than arthrotomy



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- While waiting for results of bacterial culture and antibiotic sensitivity, the veterinarian will prescribe antibiotics that kill bacteria (known as “bactericidal antibiotics”), such as first-generation cephalosporin or amoxicillin–clavulanic acid
- Choice of antibiotics (antimicrobial drugs) primarily depends on determination of antibiotic sensitivity; potential toxicity of the antibiotic, frequency of administration, route of administration, and expense also may be considered; most antibiotics penetrate the joint lining (synovium) well; antibiotics need to be given for a minimum of 4–8 weeks
- Non-steroidal anti-inflammatory drugs (NSAIDs)—may help decrease pain and inflammation; use NSAIDs only under the direction of your pet’s veterinarian



FOLLOW-UP CARE

PATIENT MONITORING

- Drainage and irrigation catheters—may be removed by your veterinarian after 4–6 days or after microscopic reassessment of joint fluid
- Duration of antibiotic therapy—2 weeks following resolution of clinical signs; total treatment may be 4–8 weeks or longer; depends on clinical signs and disease-causing organism
- Persistent joint inflammation without living bacterial organisms (dogs)—may be caused by residual antigenic bacterial fragments or antigen-antibody deposition
- Systemic steroid therapy and aggressive physical therapy—may be needed to maximize normal joint dynamics

PREVENTIONS AND AVOIDANCE

- If clinical signs recur, early (within 24–48 hours) treatment provides the greatest benefit

POSSIBLE COMPLICATIONS

- Chronic disease—severe degenerative joint disease
- Recurrence of infection
- Limited joint range of motion (stiff joint)
- Generalized infection (sepsis), involving other areas of the body
- Bone infection (osteomyelitis)

EXPECTED COURSE AND PROGNOSIS

- Acutely diagnosed disease (within 24–48 hr) responds well to antibiotic therapy
- Delayed diagnosis—guarded to poor prognosis
- The presence of bacteria or microorganisms that are resistant to antibiotics or are extremely likely to cause severe disease (virulent)—guarded to poor prognosis



KEY POINTS

- Lameness involving a single joint (monoarticular) is most common sign
- Joint pain and swelling—usually involving the carpus (joint between front paw and foreleg), stifle, hock, shoulder, or elbow joint
- Long-term antibiotic treatment is necessary
- Likelihood of residual degenerative joint disease

FLUID BUILD-UP IN THE ABDOMEN (ASCITES)



BASICS

OVERVIEW

- “Ascites” is the build-up of fluid in the abdomen

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Episodic weakness
- Sluggishness (lethargy)
- Abdominal fullness or swelling
- Discomfort when the abdomen is felt during physical examination (known as “palpation”)
- Difficulty breathing (known as “dyspnea”) from abdominal swelling, putting pressure on the chest and lungs; or associated fluid build-up in the space between the chest wall and lungs (known as “pleural effusion”)
- Lack of appetite (known as “anorexia”)
- Vomiting
- Weight gain
- Fluid build-up (known as “edema”) in the scrotum or penis
- Groaning when lying down

CAUSES

- Nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin)
- Cirrhosis (progressive damage and scarring) of the liver
- Right-sided congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)
- Low levels of protein in the blood (known as “hypoproteinemia”)
- Ruptured bladder
- Inflammation of the lining of the abdomen (known as “peritonitis”)
- Abdominal cancer
- Abdominal bleeding

RISK FACTORS

- Kidney or liver disease
- Trauma (rupture of urinary bladder; abdominal bleeding)



TREATMENT

HEALTH CARE

- Outpatient or inpatient treatment, depending on physical condition of the animal and underlying cause of fluid build-up in the abdomen (ascites)
- If patient is markedly uncomfortable when lying down or has more difficulty breathing (dyspnea) with stress, consider tapping the abdomen and removing enough fluid to reverse these signs
- Dietary salt restriction may help control some fluid accumulation related to progressive damage and scarring of the liver (cirrhosis), congestive heart failure, or low levels of protein in the blood (hypoproteinemia)
- Can re-circulate non-infected abdominal fluid in patients with liver insufficiency or nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin) that are no longer responding to conservative medical and dietary management

ACTIVITY

- Depends on underlying cause and condition of the animal

DIET

- Depends on underlying cause

- Patients with liver insufficiency or congestive heart failure—restrict sodium

SURGERY

- Corrective surgery often may be indicated (examples, to remove a tumor or to control abdominal bleeding)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Patients with liver insufficiency or congestive heart failure—medications to remove excess fluid from the body (known as “diuretics”); combination of hydrochlorothiazide and spironolactone; if control is inadequate, furosemide can be substituted for hydrochlorothiazide with spironolactone continued; must monitor serum potassium concentration to prevent potassium imbalances
- Patients with low levels of protein in the blood (hypoproteinemia), nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin), and fluid build-up in the abdomen (ascites)—treat with medications to remove excess fluid from the body (diuretics) and add colloids (fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume), such as hetastarch
- Antibiotic therapy for patients with fluid build-up in the abdomen due to bacterial infection (known as “septic ascites”); antibiotics should be selected based on bacterial culture and sensitivity testing



FOLLOW-UP CARE

PATIENT MONITORING

- Varies with the underlying cause
- Check blood work (serum chemistry profile, including sodium, potassium, blood urea nitrogen, creatinine) and body weight periodically, if the patient is maintained on medications to remove excess fluid from the body (diuretics)

PREVENTIONS AND AVOIDANCE

- Keep animals in confined locations (such as in the house or in a fenced yard) or on leash to prevent trauma

POSSIBLE COMPLICATIONS

- Aggressive administration of medications to remove excess fluid from the body (diuretics) may cause low levels of potassium in the blood (known as “hypokalemia”), which could lead to worsening of clinical signs or complications

EXPECTED COURSE AND PROGNOSIS

- Vary with the underlying cause



KEY POINTS

- “Ascites” is the build-up of fluid in the abdomen
- Aggressive administration of medications to remove excess fluid from the body (diuretics) may cause low levels of potassium in the blood (known as “hypokalemia”), which could lead to worsening of clinical signs or complications
- Keep animals in confined locations (such as in the house, in a fenced yard) or on leash to prevent trauma

DISEASE CAUSED BY *ASPERGILLUS*, A TYPE OF FUNGUS (ASPERGILLOSIS)



BASICS

OVERVIEW

- *Aspergillus* species are common molds (type of fungus) that are found throughout the environment; they form numerous spores in dust, straw, grass clippings, and hay
- “Aspergillosis” is an opportunistic fungal infection caused by a species of *Aspergillus*; “opportunistic infections” are infections caused by an organism that usually does not cause disease, but is able to cause disease because the animal’s body and/or immune system has been weakened by some other disease process
- Two types of *Aspergillus* infections—1) nasal disease, in which the infection is localized to the nasal passages and frontal sinuses and 2) widespread disease (known as “disseminated disease”); the two types do not appear to be related, but a report of a dog that developed fungal infection/inflammation of the bone marrow and bone (known as “osteomyelitis”) 6 months after treatment of the nasal type of aspergillosis raises the possibility that they may be related, at least in some animals
- Nasal disease—*Aspergillus fumigatus* most frequently involved; *A. flavus*, *A. niger*, and *A. nidulans* also isolated; presumed infection through direct introduction of the fungus into the moist lining of the nose and/or sinuses
- Widespread (disseminated) disease—usually *Aspergillus terreus*; *A. deflexus* and *A. fumigatus* also identified; portal of entry into the body not established definitively, but possibly through the respiratory tract or gastrointestinal tract, with subsequent spread through the blood stream

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Both types (nasal disease and widespread [disseminated] disease)—more common in dogs than in cats

Breed Predilections

- Nasal disease—more common in young adult dogs with a long head and nose (known as “dolichocephalic dogs,” such as the collie and Afghan hound) and dogs with a medium length head and nose (known as “mesocephalic dogs,” such as the pointer, Labrador retriever, and beagle)
- Widespread (disseminated) disease—more common in German shepherd dogs, but not confined to this breed
- Persians—marginally increased likelihood than in other cat breeds

Mean Age and Range

- Nasal disease in dogs—age range, 3 months to 11 years
- Widespread (disseminated) disease in dogs—reported average age of affected dogs, 3 years; range of 1 to 9 years of age

Predominant Sex

- Widespread (disseminated) disease in dogs—slight bias toward females being infected over males

SIGNS/OBSERVED CHANGES in the ANIMAL

Dogs

Nasal Disease

- Long-term (chronic) nasal discharge from one or both nostrils; discharge may be clear or may contain mucus, pus, and/or blood; commonly have a large volume of nasal discharge with blood and pus in it that is not responsive to treatment with antibiotics—most common sign
- Sneezing
- Nasal pain
- Bleeding from the nose and nasal passages (known as “epistaxis” or a “nosebleed”)
- Reduced appetite; sluggishness (lethargy)
- Loss of pigment (known as “depigmentation”) or loss of tissue on the surface of the skin, frequently with inflammation (known as “ulceration”) around the nostrils
- Visible distortion or swelling of the nose—uncommon
- Signs of central nervous system involvement, if the infection has moved from the nasal passages into the brain

Widespread (Disseminated) Disease

- May develop suddenly (acutely) or slowly over a period of several months
- Often associated with spinal pain due to fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition known as “fungal diskospondylitis”) or lameness due to fungal infection/inflammation of the bone marrow and bone (osteomyelitis)
- Nervous system disease—spinal cord damage
- Kidney disease—increased urination (known as “polyuria”), increased thirst (known as “polydipsia”) and blood in the urine (known as “hematuria”)
- Eye disease—inflammation of the iris and other areas in the front part of the eye (known as “uveitis”); the “iris” is the colored or pigmented part of the eye

- Nonspecific signs—fever, weight loss, vomiting, enlarged lymph nodes (known as “lymphadenopathy”), and lack of appetite (known as “anorexia”)

Cats

Nasal Disease

- Nasal discharge and noisy breathing (low-pitched, snoring sound) when inhaling (known as “stertor”)
- Frontal sinus involvement reported (with or without involvement of the bones around the eyes)

Widespread (Disseminated) Disease

- Most commonly associated with nonspecific signs (such as sluggishness [lethargy] and depression or vomiting and diarrhea)
- Eye disease—protrusion of the eyeballs (known as “exophthalmos”)
- Approximately 40 cases documented in the veterinary medical literature; majority involved widespread (disseminated) disease affecting the lungs and/or gastrointestinal tract

CAUSES

- *Aspergillus* species

RISK FACTORS

- Nasal disease—more common in outdoor dogs and farm dogs; young adult dogs with a long head and nose (dolichocephalic dogs, such as the collie and Afghan hound) and dogs with a medium length head and nose (mesaticephalic dogs, such as the pointer, Labrador retriever, and beagle)
- Widespread (disseminated) disease—German shepherd dogs most commonly affected
- Inability to produce a normal immune response (known as “immunodeficiency”)—may play a factor because *Aspergillus* species are found throughout the environment, but disease is uncommon; breed-related immune defect proposed in German shepherd dogs and their crosses
- Geographic/environmental conditions—may be a factor because some regions (such as California, Louisiana, Michigan, Georgia, Florida, and Virginia in the United States; Western Australia; Barcelona; and Milan) have a higher incidence of *Aspergillus* infection than other regions
- Cats—associated with feline infectious peritonitis (FIP), feline panleukopenia virus, feline leukemia virus (FeLV), diabetes mellitus (“sugar diabetes”), and long-term (chronic) steroid or antibiotic administration



TREATMENT

HEALTH CARE

Nasal Disease in Dogs

- Medications to kill the fungus administered by mouth or injection (known as “systemic antifungal therapy”)—limited success
- If the cribriform plate (the narrow bony structure between the nasal passages and the brain) is affected, avoid medication applied directly into the nose (known as “topical therapy”) and administer medication by mouth (known as “oral therapy”), such as itraconazole
- Clotrimazole (1%) is an antifungal drug administered directly into the nose and nasal passages, while the dog is under general anesthesia—treatment of choice; carefully monitor recovery from anesthesia following treatment with clotrimazole—some dogs may develop fluid build-up in the voice box or larynx (known as “laryngeal edema”) or in the throat (known as “pharyngeal edema”) due to irritation from the solution; more than one treatment may be required (3 to 4 treatments in some cases)
- Enilconazole is another antifungal drug that can be used instead of clotrimazole

Widespread (Disseminated) Disease in Dogs

- Difficult to eliminate infection; rare cures have been reported
- Halt progression of clinical signs, rather than eliminate infection
- Itraconazole is an antifungal drug—most effective treatment
- Combination of flucytosine and injectable amphotericin B in 4.5% saline and 2.5% dextrose—used to treat disseminated cryptococcosis (another fungal disease) successfully; may prove to have some use in treating aspergillosis (no published reports)
- Fluid therapy—indicated by the degree of kidney disease and excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”)

Cats

- Nasal disease—report of successful treatment of one cat with administration of clotrimazole (an antifungal drug) directly into the nose and nasal passages, while the cat is under general anesthesia, following treatment failure with itraconazole
- Widespread (disseminated) disease—likely difficult to treat



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Nasal Disease

- 1% clotrimazole (polyethylene glycol base preferred)—administered directly into the nasal passages (topical treatment) for 1 hour is the treatment of choice, if the cribriform plate (the narrow bony structure between the nasal passages and the brain) is intact; 90% success rate; may require more than one treatment
- Enilconazole 1% or 2% emulsion administered directly into the nasal passages for 1 hour; 80-90% success rate; may require more than one treatment
- Enilconazole—10% emulsion diluted 50:50 with water immediately before administration; administered directly into the nasal sinuses via surgically implanted frontal sinus catheters for 7 to 14 days (not appropriate as a 1-hour therapy)
- Antifungal medications administered by mouth—ketoconazole or fluconazole: much lower rates (43% to 60%) of cure than with topical treatment; itraconazole: 60% to 70% response rate reported, but cost is a factor

Widespread (Disseminated) Disease

- Itraconazole—antifungal drug of choice; dogs unlikely to be cured, though the disease may be contained with continued use
- Combination therapy with flucytosine and amphotericin B—may prove successful



FOLLOW-UP CARE

PATIENT MONITORING

- Nasal disease—nasal discharge should be well reduced 2 weeks after treatment and eliminated by 4 weeks; if still significant discharge after 2 weeks, consider a treatment failure and re-treat; consider antibiotics because bacterial infection can be a problem owing to damage sustained to lining of the nose and to the turbinates (the curved bones in the nasal passages) by the *Aspergillus* infection; recurrence of discharge after initial resolution is rarely due to recurrence of fungal infection, consider bacterial infection
- Widespread (disseminated) disease—monitor serial X-rays every 1 to 2 months, kidney function, and urine cultures

POSSIBLE COMPLICATIONS

- Depend on type of disease (nasal or widespread [disseminated])

EXPECTED COURSE AND PROGNOSIS

- Widespread (disseminated) disease in dogs—prognosis is poor, especially in the German shepherd dog



KEY POINTS

- *Aspergillus* species are common molds (type of fungus) that are found throughout the environment; they form numerous spores in dust, straw, grass clippings, and hay
- Aspergillosis is an opportunistic fungal infection caused by a species of *Aspergillus*; “opportunistic infections” are infections caused by an organism that usually does not cause disease, but is able to cause disease because the animal’s body and/or immune system has been weakened by some other disease process
- Two types of *Aspergillus* infections—1) nasal disease, in which the infection is localized to the nasal passages and frontal sinuses and 2) widespread disease (known as “disseminated disease”)

ASTHMA AND BRONCHITIS IN CATS



BASICS

OVERVIEW

- “Upper respiratory tract” (also known as the “upper airways”) includes the nose, nasal passages, throat (pharynx), and windpipe (trachea)
- “Lower respiratory tract” (also known as the “lower airways”) includes the bronchi, bronchioles, and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged)
- Asthma—sudden (acute) or long-term (chronic) inflammation of the lower airways, associated with increased responsiveness of the airways to various stimuli, airway narrowing, and presence of eosinophils, lymphocytes, and mast cells within the airways; “eosinophils” are a type of white-blood cell—they are involved in allergic responses by the body and are active in fighting larvae of parasites; “lymphocytes” are a type of white-blood cell that are formed in lymphatic tissues throughout the body—they are involved in the immune process; “mast cells” are immune-system cells that frequently are located near blood vessels—they contain histamine and are involved in allergy and inflammation
- Long-term (chronic) bronchitis—inflammation in the lower airways (bronchi and bronchioles); presents clinically as a chronic cough of greater than 2 months’ duration
- The terms “feline bronchitis” and “feline bronchopulmonary disease” are used to describe the clinical disease in cats of sudden (acute) or long-term (chronic) coughing or wheezing (squeaking or whistling sounds) accompanied by lower airway inflammation

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Breed Predispositions

- Siamese appear to be more likely to develop asthma or bronchitis than other breeds

Mean Age and Range

- Any age; more common between 2 and 8 years of age

Predominant Sex

- One study indicated that females are more likely to develop asthma or bronchitis than males; however, this is not a consistent finding

SIGNS/OBSERVED CHANGES in the ANIMAL

- Coughing (80%), sneezing (60%), labored breathing or wheezing (40%)
- Signs typically are episodic and can be sudden (acute) or long-term (chronic)
- Sluggishness (lethargy) and lack of appetite are reported occasionally
- Severely affected cats may have open-mouth breathing, rapid breathing (known as “tachypnea”), and bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body, caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”)
- Increased sensitivity to feeling the windpipe (trachea) is common
- Listening to the chest with a stethoscope (known as “chest auscultation”) may reveal short, rough snapping sounds (known as “crackles”) and/or squeaking or whistling sounds (wheezes), or may be normal
- Heart rate is typically normal to slow, although stress may result in a rapid heart rate (known as “tachycardia”)

CAUSES

- Triggers or causes of airway inflammation are largely unknown

RISK FACTORS

- Exposure to cigarette smoke, dusty cat litter, hair sprays, and air fresheners could possibly make the disease worse in some cats
- Parasitic lung infections are more common in outdoor cats in certain geographic locations
- Use of potassium bromide (treatment for seizures) has been implicated as a cause for signs of asthma or bronchitis in some cats



TREATMENT

HEALTH CARE

- Removal of cat from the inciting environment may help
- Patient should be hospitalized for a sudden (acute) crisis, characterized by breathing distress
- Oxygen therapy and sedatives may help in a sudden (acute) crisis
- Minimize handling during a crisis, in order to lessen stress and oxygen needs of the cat

ACTIVITY

- Usually self-limited by the cat

DIET

- Calorie restriction for obese cats



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Emergency Treatment

- Combine the use of oxygen and a medication that enlarges the bronchi and bronchioles in the lungs, administered by injection (medication known as a “parenteral bronchodilator”)
- A short-acting steroid, administered by injection, also may be required
- Terbutaline is a β -agonist that is used as a bronchodilator; administered by injection
- Steroids, such as dexamethasone sodium phosphate or prednisolone sodium succinate (Solu-Delta-Cortef®), administered by injection, also can be used

Long-term Management

Steroids

- Decrease inflammation
- Steroid administered by mouth (oral treatment) is preferred over administration by injection, because doses and duration can be monitored more closely; steroids include prednisone and dexamethasone (used mainly for sudden crisis); administer steroids as directed by your pet’s veterinarian
- Some cats only need steroids on a seasonal basis
- Longer-acting injectable steroids (such as Vetalog® or Depomedrol®) should be reserved for situations in which owners are unable to administer medication by mouth on a routine basis
- Inhaled steroids—newer therapy; requires a form-fitting face mask, spacer, and metered-dose inhaler; veterinary brands include AeroKat® (Trudell Medical International) or NebulAir™ (DVM Pharmaceuticals); the most commonly used inhaled steroid in cats is fluticasone propionate (Flovent®); Flovent® is used for long-term control of airway inflammation (takes 10 to 14 days to reach peak effect; during this time steroids administered by mouth should be used as well)

Other Drugs

- Medications to enlarge or dilate the bronchi and bronchioles (bronchodilators), such as theophylline and aminophylline
- β -agonists (terbutaline, albuterol)—inhibit smooth muscle constriction to dilate the bronchi and bronchioles; injectable terbutaline is most helpful in a distressed animal
- Inhaled medications to enlarge or dilate the bronchi and bronchioles (bronchodilators)—albuterol is the preferred inhalant therapy in cats, providing immediate relief of spasm and constriction of the bronchi—its effect lasts less than 4 hours
- Medications to kill intestinal parasites (known as “anthelmintics”)—routinely recommended for cats with clinical signs of feline bronchopulmonary disease, with predominantly eosinophils found on microscopic examination of material obtained from the lower airways; parasitic inflammation of the bronchi and bronchioles (bronchitis) can be difficult to diagnose—treatment is indicated with appropriate clinical signs and geographic location; appropriate medication will depend on specific parasite suspected in the geographic region; examples include fenbendazole, ivermectin, or praziquantel
- Antibiotics—choice of antibiotic based on bacterial culture and sensitivity testing
- Cyproheptadine—has been shown to inhibit airway smooth muscle constriction in some cats with asthma; should be used only in patients that do not respond to other therapy
- Cyclosporine (Neoral®)—decreases the immune response; may be helpful in cats that do not respond to medications to enlarge or dilate the bronchi and bronchioles (bronchodilators) and steroid treatment



FOLLOW-UP CARE

PATIENT MONITORING

- Watch for and report any increase in coughing, sneezing, wheezing, or breathing distress; medications should be increased appropriately if clinical signs recur, as directed by your pet’s veterinarian
- Follow-up X-rays are helpful in the first weeks after initial diagnosis to evaluate improvement with medical treatment
- Long-term use of steroids will require blood glucose monitoring every 3 to 6 months to screen for diabetes mellitus (“sugar diabetes”)
- Monitor urine (urinalysis, bacterial culture and sensitivity) as urinary tract infections can occur secondary to decreased ability to develop a normal immune response (known as “immunosuppression”)
- Watch for signs of increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”) that may indicate diabetes mellitus or kidney disease

PREVENTIONS AND AVOIDANCE

- Eliminate any environmental factors (such as cigarette smoke, dusty cat litter, hair sprays, and air fresheners) that may trigger a crisis situation
- Change furnace and air-conditioner filters on a regular basis

POSSIBLE COMPLICATIONS

- Cases that do not respond to medical treatment may die
- Untreated sudden (acute) episodes can be life threatening
- Right-sided heart disease may develop as a result of long-term inflammation of the bronchi and bronchioles (bronchitis)

EXPECTED COURSE AND PROGNOSIS

- Long-term therapy should be expected
- Most cats do well, if recurrence of clinical signs is monitored carefully and medical therapy is adjusted appropriately
- A few cats will not respond to medical treatment; these cats carry a much worse prognosis



KEY POINTS

- Inflammation of the bronchi and bronchioles (bronchitis) is a long-term (chronic), progressive diseases
- Do not discontinue medical treatment when clinical signs have resolved—subclinical inflammation within the lungs is common and can lead to progression of disease
- Life-long medication and environmental changes may be necessary, as directed by your pet's veterinarian

“DRUNKEN” APPEARING GAIT OR MOVEMENT (ATAXIA)



BASICS

OVERVIEW

- Incoordinated or “drunken” appearing gait or movement; incoordination of the limbs, head, and/or trunk
- Three clinical types of ataxia may be seen, based on location of nervous system abnormality: 1) sensory or proprioceptive abnormality in which the normal subconscious awareness of the location of the limbs and movement is altered (known as a “proprioceptive disorder”); 2) inner ear or brain-stem abnormality in which the animal’s sense of balance is altered (known as a “vestibular disorder”); and 3) abnormality of part of the brain, the cerebellum, in which limb movements are not coordinated and may be exaggerated and tremors of the head are present (known as a “cerebellar disorder”); all clinical types produce changes in limb coordination, but vestibular and cerebellar ataxia also produce changes in head and neck movement
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- “Drunken” appearing gait or movement
- Abnormal movements of the eyes
- Wide stance
- Head tilt—vestibular disorder
- Falling or rolling—vestibular disorder
- “Goose-stepping”—cerebellar disorder

CAUSES

Nervous System Causes

Spinal Cord (leading to a sensory or proprioceptive disorder)

- Degenerative disorder—loss of nervous tissue in the spinal cord (known as “degenerative radiculomyelopathy”) in old German shepherd dogs
- Disorders involving blood vessels (vascular disorders)—condition in which a piece of cartilage breaks off the intervertebral disk and travels in a blood vessel until it blocks blood flow to the spinal cord (known as “fibrocartilaginous embolic myelopathy”)
- Structural abnormalities of the spine (vertebrae) and spinal cord
- Cancer or tumors—multiple myeloma and cancer that spreads (known as “metastasis”) to the spine; primary bone tumors
- Infectious—bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition known as “diskospondylitis”); inflammation of the spinal cord (known as “myelitis”)
- Trauma to the spinal cord—intervertebral disk disease; fracture or luxation of the spine

Vestibular—Peripheral Nervous System (involving the inner ear)

- Infectious disease—middle or inner ear infection (known as “otitis media/interna”); *Cryptococcus* granuloma in cats
- Unknown cause (so called “idiopathic disease”)—geriatric vestibular disease in dogs; idiopathic vestibular syndrome in cats; inflammatory masses that develop from the middle ear or eustachian tube (known as “nasopharyngeal polyps”) in cats
- Metabolic—inadequate levels of thyroid hormone (known as “hypothyroidism”)
- Cancer or tumors—squamous cell carcinoma, bone tumors
- Trauma

Vestibular—Central Nervous System (involving the brain-stem)

- Infectious disease—feline infectious peritonitis (FIP); canine distemper virus; rickettsial diseases
- Inflammatory, idiopathic (unknown cause), or immune-mediated disorders
- Toxic—metronidazole

Cerebellar

- Degenerative—loss of nervous tissue in the cerebellum (dogs—Kerry blue terriers, Gordon setters, rough-coated collies, Australian kelpies, Airedale terriers, Bernese mountain dogs, Finnish harriers, Brittans, border collies, beagles, Samoyeds, wire fox terriers, Labrador retrievers, Great Danes, chow chows, Rhodesian ridgebacks; cats—domestic shorthairs); diseases in which normal body metabolism for certain compounds is altered (known as “storage diseases”) often have involvement of the cerebellum
- Structural abnormalities of the cerebellum—underdevelopment of the cerebellum (known as “cerebellar hypoplasia”) secondary to perinatal infection with panleukopenia virus (cats); malformed cerebellum due to herpesvirus infection (newborn puppies); arachnoid or epidermoid cyst located near fourth ventricle

- Tumor or cancer—any tumor of the central nervous system (primary or secondary) localized to the cerebellum
- Infectious disease—canine distemper virus; feline infectious peritonitis (FIP); and any other central nervous system infection affecting the cerebellum
- Inflammatory, idiopathic (unknown cause), or immune-mediated disorders
- Toxic—metronidazole

Metabolic Causes

- Low red-blood cell count (known as “anemia”)
- Electrolyte disturbances—especially low levels of potassium in the blood (known as “hypokalemia”) and low levels of sugar or glucose in the blood (known as “hypoglycemia”)

Miscellaneous Causes

- Medications—acepromazine; antihistamines; anti-seizure medications
- Breathing difficulties
- Heart and circulatory difficulties

RISK FACTORS

- Intervertebral disk disease—dachshunds, poodles, cocker spaniels, and beagles
- Wobbler syndrome (also known as “cervical spondylomyelopathy”)—Doberman pinschers and Great Danes
- Fibrocartilaginous embolism (condition in which a piece of cartilage breaks off the intervertebral disk and travels in the blood vessel until it blocks blood flow to the spinal cord)—young, large-breed dogs and miniature schnauzers
- Underdevelopment of the dens, part of the second cervical vertebra (known as “dens hypoplasia”) and dislocation of the joint between the first and second cervical vertebra (condition known as “atlantoaxial luxation”)—small-breed dogs, poodles



TREATMENT

HEALTH CARE

- Usually outpatient, depending on the severity and sudden (acute) onset of clinical signs
- Avoid drugs that could be contributing to the problem; may not be possible in patients on medications to control seizures

ACTIVITY

- Exercise—decrease or restrict if spinal cord disease is suspected



MEDICATIONS

- Not recommended until the source or cause of the problem is identified



FOLLOW-UP CARE

PATIENT MONITORING

- Periodic examinations (including the nervous system) to assess condition
- Monitor gait for increasing dysfunction or weakness; if gait or weakness worsens or paralysis develops, other testing is warranted

PREVENTIONS AND AVOIDANCE

- Avoid drugs that could be contributing to the problem; may not be possible in patients on medications to control seizures

POSSIBLE COMPLICATIONS

- Spinal cord—progression to weakness and possibly paralysis
- Low blood sugar (hypoglycemia)—seizures
- Cerebellar disease—head tremors and bobbing
- Brain-stem disease—stupor, coma, death

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause



KEY POINTS

- Incoordinated or “drunken” appearing gait or movement; incoordination of the limbs, head, and/or trunk
- Three clinical types of ataxia may be seen, based on location of nervous system abnormality: 1) sensory or proprioceptive abnormality in which the normal subconscious awareness of the location of the limbs and movement is altered (known as a “proprioceptive disorder”); 2) inner ear or brain-stem abnormality in which the animal’s sense of balance is altered (known as a “vestibular disorder”); and 3) abnormality of part of the brain, the cerebellum, in which limb movements are not coordinated and may be exaggerated and tremors of the head are present (known as a “cerebellar disorder”); all clinical types produce changes in limb coordination, but vestibular and cerebellar ataxia also produce changes in head and neck movement
- Avoid drugs that could be contributing to the problem; may not be possible in patients on medications to control seizures

ATRIAL FIBRILLATION AND ATRIAL FLUTTER



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles
- “Atrial” refers to the atrium (singular) or atria (plural) of the heart; “fibrillation” is very rapid contraction or twitching of heart muscle fibers, but not the entire muscle; “flutter” is rapid contraction of the heart muscle of the atria
- Atrial fibrillation—rapid, irregular heart rhythm involving the top two chambers of the heart (atria); two forms are recognized: 1) primary atrial fibrillation, an uncommon disease that occurs mostly in large dogs with no underlying heart disease, and 2) secondary atrial fibrillation, which occurs in dogs and cats secondary to underlying heart disease
- Atrial flutter is similar to atrial fibrillation, but the atrial rate is generally more rapid and is characterized by a regular pattern of saw-toothed flutter waves in the baseline of the electrocardiogram (“ECG,” a recording of the electrical activity of the heart); the two lower chambers of the heart (ventricles) respond to the rapid atrial heart rate; the ventricular response is generally rapid, but may be regular or irregular

GENETICS

- No breeding studies available

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Large- and giant-breed dogs are more prone to primary atrial fibrillation than are other breeds

SIGNS/OBSERVED CHANGES in the ANIMAL

- Generally relate to the underlying disease process and/or congestive heart failure (CHF) rather than the irregular heart rhythm (known as an “arrhythmia”) itself
- Dogs with primary atrial fibrillation generally are asymptomatic, but may have mild exercise intolerance
- Coughing; difficulty breathing (known as “dyspnea”); rapid breathing (known as “tachypnea”)
- Exercise intolerance
- Rarely fainting (known as “syncope”)
- Patients with atrial fibrillation have an erratic heart rhythm that sounds like “tennis shoes in a dryer” when listening to the chest with a stethoscope (procedure known as “auscultation”)
- Heart sounds when listening to the heart with a stethoscope sound like “Lub Dub;” the first heart sound is the “Lub” and the second heart sound is the “Dub;” first heart sound intensity in atrial fibrillation is variable; second heart sound only heard on some beats, not on every beat
- Third heart sounds (known as “gallop sounds”) may be present
- Patients with atrial fibrillation have pulse deficits and variable pulse quality; the “pulse” is the rhythmic “throbbing” of the arteries as the heart beats—normally the artery “throbs” each time the heart beats so that the pulse and the heart rate are the same; pulse deficits occur when the pulse and heart rate do not match, with the number of pulses being lower than the number of heart beats—pulse deficits usually indicate serious disease as the heart is unable to pump adequate blood with each heart beat; “pulse quality” is a description of how the pulse feels—words used to describe pulse quality include “weak,” “normal,” and “bounding”
- Signs of congestive heart failure (CHF) often are present; CHF signs include cough; difficulty breathing (dyspnea); bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

CAUSES

- Long-term (chronic) disease of the heart valves
- Disease of the heart muscle (known as “cardiomyopathy”)
- Congenital (present at birth) heart disease
- Digoxin toxicity; digoxin is a drug used in the treatment of some forms of heart disease
- Unknown cause (so called “idiopathic” disease)

RISK FACTORS

- Heart disease



TREATMENT

HEALTH CARE

- Consider quinidine or application of an electrical shock to the chest (known as “electrical cardioversion”) to attempt to return the heart to normal rhythm for a dog with primary atrial fibrillation; the success rate is dependent on how long the atrial fibrillation has been present—patients that have been in atrial fibrillation for more than 4 months generally have a lower success rate and a higher rate of recurrence; additional medical treatment may be necessary
- Electric shock to the chest (electrical cardioversion) to attempt to return the heart to a normal rhythm requires special equipment, trained personnel, and general anesthesia
- Patients with fast (secondary) atrial fibrillation are treated medically to slow the ventricular rate; converting the atrial fibrillation to a normal rhythm would be ideal, but such attempts in patients with severe underlying heart disease or left atrial enlargement are generally futile because of a low success rate and high rate of recurrence
- If the animal is in congestive heart failure (CHF), treatment directed at the CHF will be necessary; such treatment may include diuretics to remove excess fluid from the body, such as furosemide or spironolactone; various heart medications to control heart rate and function, such as digoxin or diltiazem; and angiotensin-converting enzyme inhibitors (ACE inhibitors) to dilate blood vessels, such as enalapril or benazepril

ACTIVITY

- Restrict activity until the rapid heart rate (known as “tachycardia”) is controlled

DIET

- Mild to moderate sodium (salt) restriction if animal is in congestive heart failure (CHF)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Digoxin, β -adrenergic blockers, and calcium channel blockers (such as diltiazem) frequently are used to slow heart rate

Dogs

- Digoxin—if digoxin is administered alone and the heart rate remains high, the veterinarian will check the digoxin level and adjust the dose; if the heart rate still remains high, the veterinarian may consider adding a calcium channel blocker or a β -adrenergic blocker
- Diltiazem
- High-dose oral quinidine can be used to convert primary atrial fibrillation into normal rhythm

Cats

- Diltiazem or atenolol are the drugs of choice in most cats
- If the heart rate is not slowed sufficiently with these drugs or if heart failure is present, digoxin can be added
- Propranolol may be used in some cases



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor heart rate and electrocardiogram (ECG; a recording of the electrical activity of the heart) closely
- Heart rates in the hospital and those measured on the ECG may be inaccurate due to patient anxiety and other environmental factors; therefore, Holter monitoring (where the patient wears a “vest” in which a continuous, mobile battery-powered ECG monitor has been placed; the ECG recording is performed over several hours, giving a better overall picture of the heart rate and rhythm) provides a more accurate means for assessing the need for heart-rate control and/or the efficacy of medical therapy for heart-rate control

POSSIBLE COMPLICATIONS

- Worsening of heart function with onset of irregular heart beats (arrhythmia)

EXPECTED COURSE AND PROGNOSIS

- Primary atrial fibrillation with normal ultrasound evaluation of the heart—generally a good prognosis
- Secondary atrial fibrillation—associated with severe heart disease, so a guarded-to-poor prognosis



KEY POINTS

- Atrial fibrillation—rapid, irregular rhythm involving the top two chambers of the heart (atria); two forms are recognized: 1) primary atrial fibrillation, an uncommon disease that occurs mostly in large dogs with no underlying heart disease, and 2) secondary atrial fibrillation, which occurs in dogs and cats secondary to underlying heart disease
- Secondary atrial fibrillation is usually associated with severe underlying heart disease; goal of therapy is to lower heart rate and control clinical signs
- Sustained conversion to a normal heart rhythm is unlikely with secondary atrial fibrillation

ATRIAL PREMATURE COMPLEXES



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- The normal heart rate for dogs varies based on the size of the dog; however, the general range is 60 to 180 beats per minute (with smaller dogs have faster normal heart rates)
- The general range for normal heart rate in cats is 120 to 240 beats per minute
- An electrocardiogram (“ECG”) is a recording of the electrical impulse activity of the heart; the normal ECG is a tracing with P, QRS, and T waves; the P waves are the first upward deflection of the ECG tracing that look like a “bump” in the tracing; the P waves are a measure of the electrical activity of the atria; the QRS looks like an exaggerated “W” with the Q wave being a short, downward deflection, the R being a tall, spiked upward deflection, and the S being another short, downward deflection; the QRS is a measure of the electrical activity of the ventricles; finally the T wave may be an upward or downward deflection of the ECG tracing; the T wave is a measure of ventricular recovery prior to the next contraction
- “Atrial premature complexes” are premature atrial beats (seen on the ECG as early or premature P waves) that originate outside the sinoatrial node and disrupt the normal heart rhythm for one or more beats
- Also known as “APCs”

ECG Features

- Heart rate usually is normal; rhythm irregular due to the early or premature P waves
- QRS complex—early or premature; appearance is usually normal

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Small-breed dogs

Mean Age and Range

- Senior animals, except those with congenital (present at birth) heart disease

SIGNS/OBSERVED CHANGES in the ANIMAL

- No signs may be seen
- Congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)
- Coughing and difficulty breathing (known as “dyspnea”)
- Exercise intolerance
- Fainting (known as “syncope”)
- Irregular heart rhythm
- Heart murmur
- Sequence of three heart sounds (known as a “gallop rhythm”), when listening to the heart with a stethoscope; heart beat sounds like a galloping horse instead of normal “lub-dub”

CAUSES AND RISK FACTORS

- Long-term (chronic) disease of the heart valve(s)
- Congenital (present at birth) heart disease
- Disease of the heart muscle (known as “cardiomyopathy”)
- Inflammation of the atrial heart muscle (known as “atrial myocarditis”)
- Electrolyte disorders
- Cancer
- Excessive levels of thyroid hormone (known as “hyperthyroidism”)
- Toxic substances in the blood (known as “toxemias”)
- Drug toxicity (such as from digitalis)
- Normal variation in aged animals



TREATMENT

HEALTH CARE

- Treat animal as inpatient or outpatient, depending on severity of clinical signs
- Treat underlying congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body's needs), heart disease, or other underlying causes
- Correct any electrolyte or acid/base imbalances

ACTIVITY

- Restrict, if animal has clinical signs

DIET

- No modifications, unless required for management of underlying condition (for example, a low-salt diet)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Various heart medications (such as digoxin, diltiazem, propranolol, or atenolol) may be used to treat clinically significant irregular heart beats (known as “arrhythmias”)
- Digoxin—treatment of choice
- Congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body's needs) is treated with medications to remove excess fluid build-up from the body (known as “diuretics”) and angiotensin-converting enzyme (ACE) inhibitors; management of congestive heart failure may reduce frequency of atrial premature complexes

Cats

- Cats with hypertrophic cardiomyopathy (disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle)—diltiazem or atenolol
- Cats with dilated cardiomyopathy (disease characterized by weak, flabby heart muscle)—digoxin



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor heart rate and rhythm with serial electrocardiograms (“ECGs,” recordings of the electrical activity of the heart)

POSSIBLE COMPLICATIONS

- Frequent atrial premature complexes may further diminish blood volume being pumped by the heart (known as “cardiac output”) in patients with underlying heart disease and worsen clinical signs

EXPECTED COURSE AND PROGNOSIS

- Even with optimal treatment using medications to control irregular heart beats (known as “anti-arrhythmic drug therapy”), some animals have an increased frequency of atrial premature complexes or deteriorate to more severe irregular heart beats (arrhythmias) as the underlying disease progresses



KEY POINTS

- Atrial premature complexes may not cause abnormalities in blood circulation; however, they may be precursors of serious irregular heart beats (arrhythmias)

ATRIAL STANDSTILL



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles
- “Atrial” refers to the atrium (singular) or atria (plural) of the heart
- “Atrial standstill” is an abnormal heart rhythm seen on a recording of an electrocardiogram (“ECG,” a recording of the electrical activity of the heart), characterized by absence of P waves—the P wave is the first upward deflection of the ECG tracing that looks like a “bump” in the tracing; the P wave is a measure of the electrical activity of the atria—absence of P waves indicates an absence of atrial activity; atrial standstill can be temporary (such as associated with too high levels of potassium in the blood [known as “hyperkalemia”] or induced by medications); persistent; or terminal (such as associated with severely high levels of potassium in the blood [severe hyperkalemia] or dying heart)
- Rare heart rhythm disturbance

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog and cat

Breed Predisposition

- Persistent atrial standstill—most common in English springer spaniels; other breeds occasionally affected

Mean Age and Range

- Most animals with persistent atrial standstill are young
- Animals with inadequate production of steroids by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”) leading to too high levels of potassium in the blood (hyperkalemia) are usually young to middle-aged

Predominant Sex

- Inadequate production of steroids by the adrenal glands (hypoadrenocorticism or Addison’s disease) more common in females (69%)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary with underlying cause
- Sluggishness (lethargy) is common; fainting (known as “syncope”) may occur
- Patients with persistent atrial standstill may show signs of congestive heart failure (CHF); CHF signs include cough; difficulty breathing (dyspnea); bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Slow heart rate (known as “bradycardia”) is common
- Patients with persistent atrial standstill may have skeletal muscle wasting of the forearms and shoulders

CAUSES

- Too high levels of potassium in the blood (hyperkalemia)
- Heart disease involving the upper chambers of the heart (known as “atrial disease”), often associated with dilated or distended atrial (such as cats with disease of the heart muscle [known as “cardiomyopathy”])
- Muscle disorder of the atria (known as “atrial myopathy”)—persistent atrial standstill

RISK FACTORS

Atrial Standstill Due to Too High Levels of Potassium in the Blood (Hyperkalemia)

- Inadequate production of steroids by the adrenal glands (hypoadrenocorticism or Addison’s disease) leading to increased levels of potassium in the blood
- Conditions leading to obstruction or rupture of the urinary tract
- Kidney failure characterized by production of only small amounts of urine (known as “oliguria”) or no urine (known as “anuria”)



TREATMENT

HEALTH CARE

Persistent Atrial Standstill

- Not life-threatening condition; animal can be treated as an outpatient

Atrial Standstill Due to Too High Levels of Potassium in the Blood (Hyperkalemia)

- Potentially life-threatening; often requires aggressive treatment
- Aggressive fluid therapy with 0.9% saline often necessary to correct low circulating blood volume (known as “hypovolemia”) and to lower serum potassium levels in patients with atrial standstill due to too high levels of potassium in the blood (hyperkalemia)

ACTIVITY

- Restrict activity in patients with persistent atrial standstill and signs of congestive heart failure (CHF) or fainting (syncope); CHF signs include cough; difficulty breathing (dyspnea); bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

SURGERY

Persistent Atrial Standstill

- Surgically implant a permanent ventricular pacemaker to regulate heart rate and rhythm

Atrial Standstill Due to Too High Levels of Potassium in the Blood (Hyperkalemia)

- Too high levels of potassium in the blood (hyperkalemia) secondary to urinary tract obstruction or rupture may require surgery to treat the problems in the urinary tract



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Persistent Atrial Standstill

- Treat with diuretics to remove excess fluid from the body (such as furosemide or spironolactone) and angiotensin-converting enzyme inhibitors (ACE inhibitors) to dilate blood vessels (such as enalapril or benazepril), if congestive heart failure (CHF) develops

Atrial Standstill Due to Too High Levels of Potassium in the Blood (Hyperkalemia)

- Treat the underlying cause (such as kidney failure characterized by production of only small amounts of urine [oliguria], inadequate production of steroids by the adrenal glands [hypoadrenocorticism or Addison’s disease])
- Aggressive fluid therapy with 0.9% saline and possibly sodium bicarbonate or insulin with dextrose
- Calcium gluconate—counters the heart effects of too high levels of potassium in the blood (hyperkalemia); can be used in life-threatening situations to reestablish a normal heart rhythm while instituting treatment to lower potassium concentration in the blood



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor electrocardiogram (ECG, a recording of the electrical activity of the heart) during treatment of too high levels of potassium in the blood (hyperkalemia) and periodically in animals with a permanent ventricular pacemaker
- Monitor electrolytes in patients with atrial standstill due to too high levels of potassium in the blood (hyperkalemia)
- Monitor patients with persistent atrial standstill for signs of congestive heart failure (CHF); CHF signs include cough; difficulty breathing (dyspnea); bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

POSSIBLE COMPLICATIONS

- Congestive heart failure (CHF) in patients with persistent atrial standstill

EXPECTED COURSE AND PROGNOSIS

Persistent Atrial Standstill

- Clinical signs generally improve after pacemaker implantation
- Signs of congestive heart failure (CHF) may develop, and weakness and sluggishness (lethargy) may persist even after heart rate and rhythm are corrected with the pacemaker; CHF signs include cough; difficulty breathing (dyspnea); bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

Atrial Standstill Due to Too High Levels of Potassium in the Blood (Hyperkalemia)

- Long-term prognosis is excellent if underlying cause can be corrected and the increased levels of potassium in the blood (hyperkalemia) are reversed



KEY POINTS

Persistent Atrial Standstill

- Clinical signs generally improve after pacemaker implantation
- Signs of congestive heart failure (CHF) may develop, and weakness and sluggishness (lethargy) may persist even after heart rate and rhythm are corrected with the pacemaker; CHF signs include cough; difficulty breathing (dyspnea); bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

Atrial Standstill Due to Too High Levels of Potassium in the Blood (Hyperkalemia)

- Potentially life-threatening; often requires aggressive treatment

FIRST-DEGREE HEART BLOCK (FIRST-DEGREE ATRIOVENTRICULAR BLOCK)



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- The normal heart rate for dogs varies based on the size of the dog; however, the general range is 60 to 180 beats per minute (with smaller dogs have faster normal heart rates)
- The general range for normal heart rate in cats is 120 to 240 beats per minute
- An electrocardiogram (“ECG”) is a recording of the electrical impulse activity of the heart; the normal ECG is a tracing with P, QRS, and T waves; the P waves are the first upward deflection of the ECG tracing that look like a “bump” in the tracing; the P waves are a measure of the electrical activity of the atria; the QRS looks like an exaggerated “W” with the Q wave being a short, downward deflection, the R wave being a tall, spiked upward deflection, and the S wave being another short, downward deflection; the QRS is a measure of the electrical activity of the ventricles; finally the T wave may be an upward or downward deflection of the ECG tracing; the T wave is a measure of ventricular recovery prior to the next contraction
- “First-degree heart block” or “first-degree atrioventricular block” refers to a delay in conduction that occurs between atrial and ventricular activation

ECG Features

- Heart rate and rhythm—usually are normal
- Regularly occurring normal P waves and QRS complexes
- Prolonged, consistent time between the P wave and the R wave (known as the “PR interval”)—dogs, greater than 0.13 sec; cats, greater than 0.09 sec

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- American cocker spaniels, dachshunds

Mean Age and Range

- May occur in young, otherwise healthy dogs as a manifestation of high vagal tone; “high vagal tone” refers to the vagus nerve—the vagus nerve provides nervous stimulation to the heart, lungs, throat, voice box, windpipe, and gastrointestinal tract; when it is stimulated (known as “vagal tone”), it has various functions, including slowing the heart
- Intra-atrial conduction delay involving the right atrium may be seen with congenital (present at birth) heart defects or disease
- May be noted in aged patients with deterioration of the electrical impulse conduction system (known as “degenerative conduction system disease”), particularly cocker spaniels and dachshunds

SIGNS/OBSERVED CHANGES in the ANIMAL

- Most animals do not have clinical signs
- If drug-induced first-degree heart block, may see signs of drug toxicity—lack of appetite (known as “anorexia”), vomiting, and diarrhea with digoxin; weakness with calcium channel blockers or β -adrenergic antagonists
- May have signs of more generalized heart muscle disease or other disease

CAUSES

- May occur in normal animals
- Increased stimulation of the vagus nerve resulting from non-heart diseases; the “vagus nerve” provides nervous stimulation to the heart, lungs, throat, voice box, windpipe, and gastrointestinal tract; when it is stimulated, it has various functions, including slowing the heart
- Medications (such as digoxin, β -adrenergic antagonists, calcium channel-blocking agents, propafenone, amiodarone, α_2 -adrenergic agonists, or severe procainamide or quinidine toxicity)
- Deterioration or degenerative disease of the conduction system
- Hypertrophic cardiomyopathy (disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle)
- Inflammation of the heart muscle (known as “myocarditis”), especially caused by infectious agents (*Trypanosoma cruzi* [Chagas’ disease], *Borrelia burgdorferi* [Lyme disease], *Rickettsia rickettsii* [Rocky Mountain spotted fever])

- Heart-muscle disease caused by infiltration with abnormal substance or cancer (known as “infiltrative cardiomyopathy”); example of disease with infiltration by an abnormal substance is amyloidosis (condition in which insoluble proteins [amyloid] are deposited outside the cells in the heart and various organs, compromising their normal function)
- Atropine administered intravenously may briefly prolong the PR interval; atropine is used as a preanesthetic medication, as an antidote, and to treat some forms of slow heart rate (known as “bradycardia”)

RISK FACTORS

- Any condition or procedure that raises vagal tone; “vagal tone” refers to the vagus nerve—the vagus nerve provides nervous stimulation to the heart, lungs, throat, voice box, windpipe, and gastrointestinal tract; when it is stimulated, it has various functions, including slowing the heart



TREATMENT

HEALTH CARE

- Remove or treat underlying cause(s)
- Hospitalization may be necessary to manage the underlying cause (such as heart muscle disease [known as “cardiomyopathy”], gastrointestinal disease, airway disease)

ACTIVITY

- Unrestricted; unless restriction required for an underlying condition

DIET

- No modifications or restrictions unless required to manage an underlying condition (for example, a low-salt diet)

SURGERY

- None, unless necessary to manage an underlying condition



MEDICATIONS

- Medications used only if needed to manage an underlying condition



FOLLOW-UP CARE

PATIENT MONITORING

- Except in healthy young animals, monitor electrocardiogram (“ECG,” a recording of the electrical activity of the heart) to detect any progression in electrical impulse conduction disturbance

EXPECTED COURSE AND PROGNOSIS

- Depends on underlying cause
- Prognosis usually excellent, if no significant underlying disease is present



KEY POINTS

- “First-degree heart block” or “first-degree atrioventricular block” refers to a delay in conduction that occurs between atrial and ventricular activation
- May be a normal finding or may be related to medications, heart disease, or other diseases stimulating the vagus nerve; the “vagus nerve” provides nervous stimulation to the heart, lungs, throat, voice box, windpipe, and gastrointestinal tract; when it is stimulated, it has various functions, including slowing the heart
- Except in healthy young animals, monitor electrocardiogram (“ECG,” a recording of the electrical activity of the heart) to detect any progression in electrical impulse conduction disturbance

SECOND-DEGREE HEART BLOCK (SECOND-DEGREE ATRIOVENTRICULAR BLOCK, MOBITZ TYPE I)



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- The normal heart rate for dogs varies based on the size of the dog; however, the general range is 60 to 180 beats per minute (with smaller dogs have faster normal heart rates)
- The general range for normal heart rate in cats is 120 to 240 beats per minute
- An electrocardiogram (“ECG”) is a recording of the electrical impulse activity of the heart; the normal ECG is a tracing with P, QRS, and T waves; the P waves are the first upward deflection of the ECG tracing that look like a “bump” in the tracing; the P waves are a measure of the electrical activity of the atria; the QRS looks like an exaggerated “W” with the Q wave being a short, downward deflection, the R wave being a tall, spiked upward deflection, and the S wave being another short, downward deflection; the QRS is a measure of the electrical activity of the ventricles; finally the T wave may be an upward or downward deflection of the ECG tracing; the T wave is a measure of ventricular recovery prior to the next contraction
- “Second-degree heart block” or “second-degree atrioventricular block” refers to failure of one or more P waves (but not all P waves) to be conducted—Mobitz type I second-degree heart block occurs when atrioventricular transmission progressively is delayed prior to a blocked P wave

ECG Features

- Time between the P wave and the R wave (known as the “PR interval”)—becomes progressively longer prior to the appearance of a P wave that is not followed by a QRS complex
- Heart rate and QRS complexes—usually are normal

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs; uncommon in cats

Mean Age and Range

- Usually occurs in young, otherwise healthy young dogs as a manifestation of high vagal tone; “high vagal tone” refers to the vagus nerve—the vagus nerve provides nervous stimulation to the heart, lungs, throat, voice box, windpipe, and gastrointestinal tract; when it is stimulated (known as “vagal tone”), it has various functions, including slowing the heart
- Occasionally occurs in older dogs with abnormally strong vagal tone
- Rarely noted in old dogs with deterioration of the electrical impulse conduction system (known as “degenerative conduction system disease”)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Most animals do not have clinical signs
- If drug-induced second-degree heart block (Mobitz type I), may see signs of drug toxicity—lack of appetite (known as “anorexia”), vomiting, and diarrhea with digoxin; weakness with calcium channel blockers or β -adrenergic antagonists
- If heart rate is abnormally slow, fainting (known as “syncope”) or weakness may occur
- May have signs of more generalized heart muscle disease or other disease
- May have a change in heart sounds heard when listening to the heart with a stethoscope (known as “auscultation”)

CAUSES

- Occasionally noted in normal animals
- Increased vagal stimulation resulting from non-heart diseases; the “vagus nerve” provides nervous stimulation to the heart, lungs, throat, voice box, windpipe, and gastrointestinal tract; when it is stimulated, it has various functions, including slowing the heart
- Medications (such as digoxin, β -adrenergic antagonists, calcium channel-blocking agents, propafenone, amiodarone, α_2 -adrenergic agonists, or opioids)

RISK FACTORS

- Any condition or procedure that raises vagal tone; “vagal tone” refers to the vagus nerve—the vagus nerve provides nervous stimulation to the heart, lungs, throat, voice box, windpipe, and gastrointestinal tract; when it is stimulated, it has various functions, including slowing the heart



TREATMENT

HEALTH CARE

- Treatment usually is unnecessary
- Treat or remove underlying cause(s)

ACTIVITY

- Unrestricted

DIET

- Modifications or restrictions only to manage an underlying condition

SURGERY

- None, unless necessary to manage an underlying condition



MEDICATIONS

- Only as needed to manage an underlying condition



FOLLOW-UP CARE

PATIENT MONITORING

- Typically not necessary



KEY POINTS

- Any treatment is directed toward reversing or eliminating an underlying cause

SECOND-DEGREE HEART BLOCK (SECOND-DEGREE ATRIOVENTRICULAR BLOCK, MOBITZ TYPE II)



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- The normal heart rate for dogs varies based on the size of the dog; however, the general range is 60 to 180 beats per minute (with smaller dogs have faster normal heart rates)
- The general range for normal heart rate in cats is 120 to 240 beats per minute
- An electrocardiogram (“ECG”) is a recording of the electrical impulse activity of the heart; the normal ECG is a tracing with P, QRS, and T waves; the P waves are the first upward deflection of the ECG tracing that look like a “bump” in the tracing; the P waves are a measure of the electrical activity of the atria; the QRS looks like an exaggerated “W” with the Q wave being a short, downward deflection, the R wave being a tall, spiked upward deflection, and the S wave being another short, downward deflection; the QRS is a measure of the electrical activity of the ventricles; finally the T wave may be an upward or downward deflection of the ECG tracing; the T wave is a measure of ventricular recovery prior to the next contraction
- “Second-degree heart block” or “second-degree atrioventricular block” refers to failure of one or more P waves (but not all P waves) to be conducted—Mobitz type II second-degree heart block occurs when one or more P waves are blocked, without a preceding progressive delay in atrioventricular transmission

ECG Features

- One or more P waves not followed by a QRS complex, and time between the P wave and the R wave (known as the “PR interval”) of conducted beats is consistent
- Ventricular rate—usually slow
- QRS complexes may appear normal or may be wide or have an abnormal appearance
- Abnormally wide QRS complexes may indicate serious, extensive heart disease

GENETICS

- May be inherited in pugs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- American cocker spaniels
- Pugs
- Dachshunds

Mean Age and Range

- Generally occurs in older animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Animal may have no clinical signs
- Fainting (known as “syncope”), collapse, weakness, or sluggishness (lethargy)
- May have signs of underlying disease
- Slow heart rate (known as “bradycardia”) is common
- May be intermittent pauses in the heart rhythm
- May have a change in heart sounds heard when listening to the heart with a stethoscope (known as “auscultation”)
- If associated with digoxin toxicity, may see vomiting, lack of appetite (known as “anorexia”), and diarrhea; “digoxin” is a heart medication

CAUSES

- May be inherited in pugs
- Increased stimulation of the vagus nerve resulting from non-heart diseases; the “vagus nerve” provides nervous stimulation to the heart, lungs, throat, voice box, windpipe, and gastrointestinal tract; when it is stimulated, it has various functions, including slowing the heart

- Deterioration of the electrical impulse conduction system (known as “degenerative conduction system disease”)
- Medications (such as digoxin, β -adrenergic antagonists, calcium channel-blocking agents, propafenone, amiodarone, α_2 -adrenergic agonists, muscarinic cholinergic agonists, or severe procainamide or quinidine toxicity)
- Heart-muscle disease caused by infiltration with abnormal substance or cancer (known as “infiltrative cardiomyopathy”); example of disease with infiltration by an abnormal substance is amyloidosis (condition in which insoluble proteins [amyloid] are deposited outside the cells in the heart and various organs, compromising their normal function)
- Inflammation of the lining of the heart (known as “endocarditis”), particularly involving the aortic valve
- Infection/inflammation of the heart muscle (known as “myocarditis”)—viral, bacterial and parasitic causes; unknown causes (so called “idiopathic myocarditis”)
- Disease of the heart muscle (known as “cardiomyopathy”), especially in cats
- Trauma
- Atropine administered intravenously may cause a brief period of first- or second-degree heart block before increasing the heart rate; atropine is used as a preanesthetic medication, as an antidote, and to treat some forms of slow heart rate (bradycardia)

RISK FACTORS

- Any condition or procedure that raises vagal tone; “vagal tone” refers to the vagus nerve—the vagus nerve provides nervous stimulation to the heart, lungs, throat, voice box, windpipe, and gastrointestinal tract; when it is stimulated, it has various functions, including slowing the heart



TREATMENT

HEALTH CARE

- Treatment—may be unnecessary, if heart rate maintains adequate blood volume being pumped by the heart (known as “cardiac output”)
- Medication to improve electrical impulse conduction or implantation of a pacemaker is indicated for pets having clinical signs
- Treat or remove underlying cause(s)

ACTIVITY

- Cage rest advised for pets having clinical signs

DIET

- Modifications or restrictions only to manage an underlying condition

SURGERY

- Permanent pacemaker may be required for long-term management of pets having clinical signs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Atropine or glycopyrrolate may be used short term, if animal has positive atropine response; atropine is used as a preanesthetic medication, as an antidote, and to treat some forms of slow heart rate (bradycardia); glycopyrrolate is a drug with similar effects and uses as atropine
- Long-term (chronic) anticholinergic therapy (drugs such as propantheline or hyoscyamine)—indicated for pets having clinical signs, if they had improved atrioventricular (AV) conduction with atropine response test
- Isoproterenol (drug used to control some types of irregular heart beats) or dopamine (drug used to increase contraction of heart muscle) may be administered in sudden (acute), life-threatening situations to enhance atrioventricular (AV) conduction



FOLLOW-UP CARE

PATIENT MONITORING

- Frequent electrocardiograms (“ECGs,” recordings of the electrical activity of the heart) because second-degree heart block (Mobitz type II) often progresses to complete (third-degree) heart block

POSSIBLE COMPLICATIONS

- Prolonged slow heart rate (bradycardia) may cause secondary congestive heart failure or inadequate blood flow to the kidneys; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

EXPECTED COURSE AND PROGNOSIS

- Variable—depends on underlying cause
- If animal has deterioration of the electrical impulse conduction system, second-degree heart block (Mobitz type II) often progresses to complete (third-degree) heart block



KEY POINTS

- Identify and specifically treat underlying cause
- Medications may not be effective long-term
- If animal has deterioration of the electrical impulse conduction system, second-degree heart block (Mobitz type II) often progresses to complete (third-degree) heart block
- Prolonged slow heart rate (bradycardia) may cause secondary congestive heart failure or inadequate blood flow to the kidneys; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

COMPLETE HEART BLOCK (THIRD-DEGREE ATRIOVENTRICULAR BLOCK)



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- The normal heart rate for dogs varies based on the size of the dog; however, the general range is 60 to 180 beats per minute (with smaller dogs have faster normal heart rates)
- The general range for normal heart rate in cats is 120 to 240 beats per minute
- An electrocardiogram (“ECG”) is a recording of the electrical impulse activity of the heart; the normal ECG is a tracing with P, QRS, and T waves; the P waves are the first upward deflection of the ECG tracing that look like a “bump” in the tracing; the P waves are a measure of the electrical activity of the atria; the QRS looks like an exaggerated “W” with the Q wave being a short, downward deflection, the R being a tall, spiked upward deflection, and the S being another short, downward deflection; the QRS is a measure of the electrical activity of the ventricles; finally the T wave may be an upward or downward deflection of the ECG tracing; the T wave is a measure of ventricular recovery prior to the next contraction
- “Complete heart block” or “third-degree atrioventricular block” occurs when all atrial impulses are blocked at the atrioventricular (AV) node; therefore, the atria and ventricles beat independently and are no longer beating in a coordinated fashion—a secondary “escape” pacemaker site (either near the AV node or in the ventricle) stimulates the ventricles
- The atrial rate is normal (that is, the normal pacemaker or sinoatrial node starts the electrical impulse to begin at a normal rate, which causes the atria to contract at a normal rate)
- The ventricular rate (known as an “idioventricular escape rhythm”) is slow

ECG Features

- Ventricular rate slower than the atrial rate (that is, more P waves are present than are QRS complexes)—ventricular escape rhythm (idioventricular) usually less than 40 beats per minute; junctional (near the atrioventricular node) escape rhythm (idiojunctional) 40 to 60 beats per minute in dogs and 60 to 100 beats per minute in cats
- P waves—usually normal appearance
- QRS complex—may be wide and bizarre or normal, depending on location of the secondary “escape” pacemaker
- No conduction occurs between the atria and the ventricles; P waves have no constant relationship with QRS complexes

GENETICS

- Can be an isolated congenital (present at birth) defect

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Cocker spaniels—can have scarring of heart tissue of unknown cause (so called idiopathic fibrosis)
- Pugs and Doberman pinschers—can have associated sudden death, defects in conduction of impulses from the atria to the ventricles (known as “atrioventricular conduction defects”), and lesions in the specialized heart tissue (known as the “bundle of His”) that conducts electrical impulses into the ventricles

Mean Age and Range

- Senior animals, except congenital (present at birth) heart disease patients
- Median age for cats is 14 years

Predominant Sex

- Intact female dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Exercise intolerance
- Weakness or fainting (known as “syncope”)
- Occasionally, congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)
- Slow heart rate (known as “bradycardia”)
- Change in heart sounds, when listening to the heart with a stethoscope (known as “auscultation”)
- Coughing and difficulty breathing (known as “dyspnea”)

CAUSES AND RISK FACTORS

- Isolated congenital (present at birth) defect
- Scarring of heart tissue of unknown cause (idiopathic fibrosis)
- Heart-muscle disease caused by infiltration with abnormal substance or cancer (known as “infiltrative cardiomyopathy”); example of disease with infiltration by an abnormal substance is amyloidosis (condition in which insoluble proteins [amyloid] are deposited outside the cells in the heart and various organs, compromising their normal function)
- Hypertrophic cardiomyopathy (disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle) in cats
- Digitalis toxicity; “digitalis” is a heart medication
- Inflammation of the heart muscle (known as “myocarditis”)
- Inflammation of the lining of the heart (known as “endocarditis”)
- Electrolyte disorder
- Sudden lack of blood supply to the heart muscle that leads to death of tissues (known as “myocardial infarction”)
- Other congenital (present at birth) heart defects
- Lyme disease (tick-borne disease, caused by *Borrelia burgdorferi*)
- Chagas’ disease (disease caused by a parasite, *Trypanosoma*, that damages heart muscle)



TREATMENT

HEALTH CARE

- Temporary or permanent heart pacemaker—only effective treatment in patients with clinical signs
- Cage rest prior to pacemaker implantation; when the pulse generator of the pacemaker is placed surgically into a pocket under the skin (known as a “subcutaneous pocket”), a nonconstrictive bandage is required for 3 to 5 days to prevent formation of a localized accumulation of serum (known as a “seroma”) or pacemaker movement

ACTIVITY

- Restrict, if animal has clinical signs
- Cage rest prior to pacemaker implantation

DIET

- No modifications, unless required for management of underlying condition (for example, a low-salt diet)

SURGERY

- Most patients—at high risk for undergoing surgery; heart rate usually paced preoperatively with a temporary external pacemaker system
- The small size of cats makes pacemaker implantation more difficult than in dogs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treatment with drugs—usually of no value
- Medications traditionally used to treat complete heart block (complete atrioventricular block): atropine, isoproterenol, theophylline, steroids, and dobutamine
- Intravenous isoproterenol infusion may help increase the ventricular rate to stabilize circulation of blood
- If animal has signs of congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)—medications to remove excess fluid build-up (known as “diuretics”) and to enlarge or dilate blood vessels (known as “vasodilator therapy”) may be needed before pacemaker implantation



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor—pacemaker function with serial electrocardiograms (“ECGs,” recordings of the electrical activity of the heart)

- Chest X-rays—following pacemaker implantation, to confirm the position of the lead and generator
- Carefully monitor patients that do not have clinical signs and have not had a pacemaker implanted for development of clinical signs

POSSIBLE COMPLICATIONS

- Pulse generators—broad range of clinical life; pacemaker replacement necessary when battery is depleted, pulse generator malfunction occurs, or exit block develops
- Pacemaker leads can become dislodged and infected

EXPECTED COURSE AND PROGNOSIS

- Poor long-term prognosis, if no heart pacemaker is implanted, especially when the animal has clinical signs
- Cats can sometimes survive more than one year without having a pacemaker implanted



KEY POINTS

- Temporary or permanent heart pacemaker—only effective treatment in patients with clinical signs of complete heart block (third-degree atrioventricular block)
- Patients without clinical signs and that have not had a pacemaker implanted must be monitored carefully for development of clinical signs

ATRIOVENTRICULAR VALVE DYSPLASIA



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the left and right atria and the bottom two chambers are the left and right ventricles; heart valves are located between the left atrium and the left ventricle (mitral valve); between the right atrium and the right ventricle (tricuspid valve); from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve); and from the right ventricle to the main pulmonary (lung) artery (pulmonary valve)
- The mitral valve and the tricuspid valves are the “atrioventricular valves;” that is, they are the valves between the left atrium and ventricle and between the right atrium and ventricle, respectively
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- “Dysplasia” is the medical term for abnormal development of a tissue
- “Atrioventricular valve dysplasia” is a congenital (present at birth) malformation of the mitral or tricuspid valve

GENETICS

- Tricuspid valve dysplasia is inherited as an autosomal recessive trait in Labrador retrievers
- Heritability and pattern of inheritance are not established in other breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- One of the most common congenital (present at birth) heart defects in cats; less frequently diagnosed in dogs

Breed Predispositions

- Mitral valve dysplasia—bull terriers, Newfoundlands, Great Danes, golden retrievers, possibly Dalmatians and Siamese cats; perhaps the most common congenital (present at birth) heart defect of cats
- Mitral valve malformations also common in cats with hypertrophic cardiomyopathy (disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle)
- Tricuspid valve dysplasia—Labrador retrievers, German shepherd dogs, Great Pyrenees, possibly Old English sheepdogs; common in cats

Mean Age and Range

- Variable
- Signs most often seen within the first few years of life

Predominant Sex

- Males are more likely than females to have signs of heart failure

SIGNS/OBSERVED CHANGES in the ANIMAL

- Exercise intolerance most common problem in dogs and cats
- Abdominal swelling or distention, weight loss, and stunting of growth may be observed with severe tricuspid valve dysplasia
- Labored breathing common in dogs or cats with mitral valve dysplasia
- Fainting (known as “syncope”) and collapse, if severe mitral or tricuspid valve narrowing (known as “stenosis”) or if blood flow through the valves is blocked or the animal has irregular heart beats (known as “arrhythmias”)

Mitral Valve Dysplasia (involves the valve between the left atrium and the left ventricle)

- Heart murmur; with severe disease, may be able to feel vibrations caused by abnormal blood flow (known as “thrills”) when placing hand against the chest wall or may hear a sequence of three heart sounds (known as a “gallop rhythm”), when listening to the heart with a stethoscope; heart beat sounds like a galloping horse instead of normal “lub-dub”
- Evidence of left-sided congestive heart failure—animals with severe defects may have rapid breathing (known as “tachypnea”); increased breathing efforts; rough snapping sounds (known as “crackles”) may be heard when listening to the chest with a stethoscope; and bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

Tricuspid Valve Dysplasia (involves the valve between the right atrium and the right ventricle)

- Heart murmur; with severe disease, may be able to feel vibrations caused by abnormal blood flow (thrills) when placing hand against the chest wall or may hear a sequence of three heart sounds (gallop rhythm), when listening to the heart with a stethoscope; heart beat sounds like a galloping horse instead of normal “lub-dub”
- The external jugular veins (located on either side of the neck) may be enlarged or distended and may have a pulse
- Evidence of right-sided congestive heart failure—fluid build-up in the abdomen (known as “ascites”) and, more rarely, fluid build-up in the tissues, especially the legs and under the skin (known as “peripheral edema”) with severe malformations

CAUSES

- Congenital (present at birth) malformation of the mitral or tricuspid valve



TREATMENT

HEALTH CARE

- Inpatient treatment required for congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

ACTIVITY

- Restricted in accordance with severity of clinical signs

DIET

- Sodium-restricted diet, if in or likely to develop congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

SURGERY

- Heart valve surgery is available in a few centers
- Surgical treatment is expensive



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Mitral or tricuspid dysplasia with insufficiency—medications to remove excess fluid from the body (known as “diuretics,” such as furosemide), heart medications (such as angiotensin-converting enzyme [ACE] inhibitors and digoxin) for patients in or likely to develop congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Mitral or tricuspid narrowing (stenosis)—medications to remove excess fluid from the body (diuretics, such as furosemide) to control fluid build-up (known as “edema”); heart rate should be maintained near 150 beats per minute, using digoxin, a calcium-channel blocker (such as diltiazem), or a beta-receptor blocking drug (such as atenolol)
- Dynamic blockage of blood flow through the valves (known as “dynamic outflow tract obstruction”)—beta-receptor blocking drug (such as atenolol) to decrease severity of outflow obstruction; a “dynamic” process in one in which the lumen of the chambers changes with the movements of the heart (relaxation and contraction)
- Medications to remove excess fluid from the body (diuretics, such as furosemide), if animal is in congestive heart failure



FOLLOW-UP CARE

PATIENT MONITORING

- Recheck yearly, if no signs of congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Recheck at a minimum of every 3 months, if signs of congestive heart failure—chest X-rays, electrocardiogram (“ECG,” a recording of the electrical activity of the heart), and echocardiography (use of ultrasound to evaluate the heart and major blood vessels)

PREVENTIONS AND AVOIDANCE

- Do not breed affected animals

POSSIBLE COMPLICATIONS

- Congestive heart failure: left-sided with mitral valve dysplasia; right-sided with tricuspid valve dysplasia; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Collapse or fainting (syncope) with exercise
- Sudden onset (known as “paroxysmal”) supraventricular tachycardia or atrial fibrillation with severe disease; “supraventricular tachycardia” is a rapid heart rate caused by electrical impulses that originate from a site other than the sinoatrial (SA) node, the normal pacemaker of the heart; “atrial fibrillation” is a rapid, irregular heart rhythm involving the top two chambers of the heart (atria)

EXPECTED COURSE AND PROGNOSIS

- Depends on severity of underlying defect
- Prognosis is guarded to poor with serious defects



KEY POINTS

- “Atrioventricular valve dysplasia” is a congenital (present at birth) malformation of the mitral or tricuspid valve
- Potentially an inherited disorder; affected animals should not be used for breeding

UNRULY BEHAVIOR IN DOGS: JUMPING, DIGGING, CHASING, STEALING



BASICS

OVERVIEW

- Jumping—standing on rear legs with front legs on a person or object or leaping in the air with or without landing against the person
- Digging—using paws to scrape a surface, as though attempting to excavate the underlying layer
- Chasing—pursuing a moving person, animal, or object
- Stealing—the taking of an item not intended to be utilized by the dog

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilection

- Herding and hunting breeds may be more likely to chase than other breeds
- Hunting breeds (including the terrier breeds) may be more likely to dig than other breeds

Mean Age and Range

- More common in younger dogs, but may occur at any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Jumping up on people occurs more commonly in association with arrivals or departures or greeting at other times; it also is associated with exploring the contents of countertops or tables
- Digging often occurs in areas along a fence line or areas of recent gardening, at rodent holes, and on interior flooring with or without owner presence
- Items displaced or food items missing from surfaces are common complaints in stealing
- Physical examination findings usually are unremarkable
- Nails worn down
- Pain on feeling the abdomen (known as “abdominal palpation”) may suggest an organic disease
- Nervous system examination may suggest an organic disease

CAUSES

- Jumping up is a normal greeting and play behavior; excitement, encouragement of the behavior by others, or inadvertent rewarding of the behavior perpetuates it
- Separation anxiety may result in excessive jumping on owners when returning home or leaving
- Digging is a normal behavior; presence of rodents, anxiety, regulation of body temperature, under-stimulation or lack of adequate exercise, food hiding or retrieval, escape from confinement, pain (particularly abdominal), separation anxiety, obsessive-compulsive disorder (OCD), and nervous system disease can be causes of digging
- Chasing is a normal behavior; causes include herding, hunting, play, and defense
- Stealing is a normal acquisitive behavior; it can be caused by a dog trying to get an owner’s attention or by the desire for a food item

RISK FACTORS

- Inadequate exercise
- Under-stimulation
- Stealing food—restricted or weight-reduction diets; certain medications (such as phenobarbital, benzodiazepines, and steroids); diseases (such as increased production of steroids by the adrenal glands [known as “hyperadrenocorticism” or “Cushing’s disease”] and sugar diabetes [diabetes mellitus])
- Chasing—lack of exposure to fast moving stimuli at a young age, common in herding breeds



TREATMENT

HEALTH CARE

- Outpatient management

ACTIVITY

- Increase the dog's daily exercise



FOLLOW-UP CARE

PATIENT MONITORING

- Every 2 to 3 weeks initially

PREVENTIONS AND AVOIDANCE

- Close supervision, exercise, and exposing the dog to different stimuli as a young puppy can help to prevent some of the unruly behaviors

POSSIBLE COMPLICATIONS

- Injury as a result of escaping a fence, chasing a stimulus, or ingesting an inappropriate item

EXPECTED COURSE AND PROGNOSIS

- Generally good response to treatment for jumping, digging, and stealing, if the owner is consistent in modifying the behavior
- Chasing behaviors may be more difficult and resistant to treatment



KEY POINTS

Jumping

- During training, prevention of the undesired jumping-up behavior is essential
- A head collar and leash facilitate training to gently guide the dog away from jumping
- Greeting visitors outside might diminish jumping behavior, or the dog's access to the situation can be restricted by placing it in another room until the visitor is seated
- Teach "sit" and "stay" as an alternative method to greet people
- When the dog is calm and easily controlled, practice sitting for a food reward in different areas of the house
- Sessions should be short—3 to 5 minutes with 8 to 12 repetitions per session
- Food rewards should be highly palatable and small (1/4-inch square or larger, depending on the size and weight of the dog); initially food rewards should be used consistently
- Add the word "stay" when the duration of sitting is a few seconds; take a step away, return to the dog and give the food reward—build up the time away from the dog to 3 to 5 minutes
- Repeat exercises near the door and with the addition of leaving and returning
- Next ask the dog to "sit" for a food reward when returning from work or other absences of a few hours' duration
- Familiar visitors can enter, ask the dog to "sit," and give a food reward
- Alternatively, the owner can reward the dog for remaining seated as visitors enter
- Eventually the food rewards can be reduced to intermittent, but frequent use
- Dogs that like to retrieve and are too excited to sit may do better if a ball is tossed as a visitor enters—this is more beneficial if a dog has been taught to sit prior to an item's being tossed again
- The owner should avoid increasing the dog's excitement by walking calmly to the door and speaking in a quiet voice

- People should avoid rewarding the jumping with attention (such as pushing the dog off); do not acknowledge or interact with the dog; hold arms against the body, and turn body away from the dog—some dogs will stop jumping and ignore the person
- Stepping on the dog's toes or squeezing the paws and other punishment usually are ineffective and can lead to aggression; therefore, these "techniques" should be avoided

Digging

- Digging associated with temperature regulation occurs in hot or cold weather to help cool or to conserve body heat, respectively; an adequately cooled or heated environment or shelter should minimize this problem
- Digging associated with rodents can occur inside or outside; this behavior likely will persist until the rodents are removed
- Digging associated with separation anxiety, escaping a phobic stimulus, or obsessive-compulsive disorder (OCD) should resolve with treatment of those conditions; dogs with separation anxiety should not be left alone in the yard for extended periods of time
- For digging in the owner's presence or not associated with any of the previously described situations, the owner should increase the dog's exercise and activity; aerobic exercise should occur before leaving the dog unsupervised in the yard—the provision of interactive toys (like automatic tennis-ball throwers) may be helpful
- If the digging persists, create an area where it is acceptable for the dog to dig; a children's sandbox can be used for this purpose or an area can be marked off with wood and filled with sand or topsoil; initially bury toys or food items with the dog observing, so the dog is directed to and rewarded for digging in the specific area
- Supervision is necessary to redirect the dog to another activity as it starts to dig; aversive stimuli (such as loud noise, water spray) can be used to interrupt the digging, but may not affect the digging when the owner is absent; a motion-activated sprinkler or putting stones or water in the dug area might prevent digging in that specific area, but may not affect digging in other areas, so they are not recommended

Chasing

- A no-pull harness or head collar can be helpful in controlling the dog in the presence of the chase stimulus; herding breeds exhibit a phenotypic behavior that may respond better to control and management than to treatment
- Dogs that chase will need to be exposed to (desensitized) and taught a different response (counter-conditioned) to the stimulus; this should be done gradually
- The owner should use "sit-and-stay" exercises with the dog, with the addition of a "look" command, using a treat brought up to the owner's eye—this will help get the dog's attention and focus it back on the owner, when the dog sees the moving stimulus
- Sessions should be 3 to 5 minutes, with numerous repetitions per session; initially work with the dog inside without distractions, having it "sit," "stay," and "look"
- Next work in a quiet yard with the dog on a leash, have the dog "sit," "stay," step away, return, "look," and give the food reward; when the dog is successful, the process can be repeated in a more distracting part of the yard—if the dog is too distracted, the owner should work with the dog at times of day when fewer distractions (such as passers-by or traffic) are present
- The owner should first work with the dog without the chase stimulus present; if the owner is able to keep the dog's attention, the owner then should stage the chase stimulus (like a bike or person jogging) to pass by at a great distance while working with the dog; the owner might need to increase the speed of the repetitions and rewards
- Each day, if the dog is able to ignore the chase stimulus, the owner should move a few inches closer to the chase stimulus; if the dog is unable to ignore the chase stimulus, the distance should be increased—when the dog is able to ignore the chase stimulus in the yard, the owner can incorporate the same exercise on a walk; when the owner sees the chase stimulus, he or she should ask the dog to "sit," "stay," and "look," and then reward the dog

Stealing

- The dog's attempts to initiate play and chase may result in stealing
- Adequate attention, exercise, and toys before the owner becomes preoccupied (such as making dinner, working, watching television) will help to decrease this motivation for stealing
- Owners should not engage in chasing the dog; they should ignore the dog and walk away, get a treat, and call the dog to them—while the dog is in the process of dropping the item, the owner can say "drop," "good dog," and give the dog the treat—the dog is being rewarded for relinquishing the item
- The owner may want to give a second treat so the dog does not "race" for the dropped item; the item should be placed out of view and not shown to the dog
- If the dog retreats under furniture, the owner should not pursue it—if the dog feels threatened or cornered, it may defend itself aggressively
- For food stealing, food needs to be placed out of the dog's reach, since acquiring food is highly rewarding
- Products that can interrupt the dog's behavior and are mildly aversive can help correct the stealing behavior; motion detectors can be helpful in deterring stealing behavior
- If the dog steals food because it is on a diet, a protein source (such as dried chicken breast strips) or low-calorie foods (such as raw or cooked vegetables) can be added to the dog's food so it might feel less hungry

MATERNAL BEHAVIOR PROBLEMS



BASICS

OVERVIEW

- Abnormal maternal behavior is either excessive maternal behavior in the absence of newborns or deficient maternal behavior in the presence of the dam's own newborns
- The female dog is known as the "bitch" and the female cat is known as the "queen"

GENETICS

- No genetic basis has been identified in dogs and cats, but a breed tendency in Jack Russell terriers indicates that a genetic component may be involved
- Genetic models of deficient maternal behavior in mice have been identified; the genes responsible for deficient maternal behavior in mice are imprinted paternally—if this situation is true in dogs and cats, one would expect that rejecting mothers were normally mothered themselves, but their grandmother may have been deficient
- The genetic basis should be investigated in dogs and cats

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Poor maternal behavior may be more common in Jack Russell terriers than in other breeds of dog, but no quantitative study has proven this observation

Predominant Sex

- Female only

SIGNS/OBSERVED CHANGES in the ANIMAL

Deficient Maternal Behavior

- Absent maternal behavior; the mother simply abandons her offspring—this is most apt to occur after caesarean section

Poor Maternal Behavior

- The mother stays with her offspring, but will not allow them to nurse
- The mother may show inadequate retrieval of young, insufficient cleaning of the young, or failure to stimulate elimination (urination and/or defecation) by the young
- The bitch carries the puppies from place-to-place without settling down or, in the most extreme form, kills some or all of her litter

Abnormal Maternal Behavior

- The bitch or queen may allow her offspring to suckle, but kills her offspring either at birth or over a period of days
- Occasionally the bitch, or more rarely the queen, will abandon or attack her offspring, if it has changed in odor or appearance
- A female may be disturbed by another animal or by people and can redirect her aggression to her offspring
- A bitch accidentally may disembowel or even consume offspring completely while eating the fetal membranes (placenta) and umbilical cord; this should be distinguished from normal licking, which can be quite vigorous, even to the point of dislodging the puppy from a nipple

Maternal Aggression

- Cats with kittens may be aggressive to other animals, especially dogs in the same household

Excessive Maternal Behavior

- The "pseudopregnant" bitch or bitch spayed during the latter phase of the estrous or heat cycle may show signs of a false pregnancy; she attempts to nurse, and guards inanimate objects (stuffed animals or even leashes)
- The pseudopregnant bitch may have breast or mammary development and may be producing milk (lactating)
- The newly spayed queen may steal kittens from a nursing queen; queens also may produce milk (lactate) if suckled, following the spay (ovariohysterectomy)

CAUSES

- The presence of kittens in the environment of the recently spayed cat is a risk factor for excessive maternal behavior and kitten stealing

- The risk of excessive carrying of puppies, redirected aggression, or even cannibalism is increased if other dogs or too many people are present in the nest area



TREATMENT

HEALTH CARE

- Normal health care

DIET

- Adequate diet for nursing bitches and queens to meet energy demands
- Restricted diets for animals with false pregnancies (known as “pseudocyesis”), to discourage lactation
- In the case of deficient maternal behavior, the bitch or queen should be fed free choice (known as “ad libitum”) to encourage lactation

SURGERY

- Delay spaying for four months post-estrus to avoid post-spaying maternal behavior and its accompanying aggression
- Spaying avoids future excessive maternal behavior in the absence of young



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Excessive Maternal Behavior

- Mibolerone (Cheque® Drops) is the drug of choice for bitches with false pregnancies or those exhibiting maternal behavior and lactation following spaying; mibolerone inhibits prolactin (the hormone that stimulates secretion of milk) and thereby inhibits lactation

Deficient Maternal Behavior

- Oxytocin (the hormone that stimulates milk release during nursing) may be administered either by injection or by nasal spray (Syntocinon®)
- Prolactin (the hormone that stimulates secretion of milk) appears to be involved with maternal behavior in other species; therefore, a dopamine blocker (acepromazine) can be used; dopamine inhibits prolactin release and thus a dopamine blocker would increase prolactin



FOLLOW-UP CARE

PATIENT MONITORING

- The puppies or kittens of females with deficient or poor maternal behavior should be monitored daily to be sure that they are gaining weight

PREVENTIONS AND AVOIDANCE

- Place a nursing female and her litter in quiet, comfortable quarters—away from noise and disturbances by other animals or people
- Do not re-breed females with poor maternal behavior; deficient maternal behavior can occur with each litter
- Determine whether any other female offspring of the female with abnormal maternal behavior also exhibited poor maternal behavior
- In other species, poor maternal behavior is a paternally imprinted gene; the father must contribute the gene for poor maternal behavior; the daughters of rejecting mothers will not reject, but daughters of their sons may have poor maternal behavior

POSSIBLE COMPLICATIONS

- Loss of offspring

EXPECTED COURSE AND PROGNOSIS

- Excessive maternal behavior usually wanes around the time of normal weaning (6 to 8 weeks)

- Poor and deficient maternal behavior can occur with each litter



KEY POINTS

Abnormal or Poor Maternal Behavior

- The bitch that is carrying her puppies or exhibiting redirected aggression to them should be isolated in a quiet, dark area
- The bitch that bites her puppies should be muzzled; the owner must stimulate elimination (urination and/or defecation) of the puppies, because the muzzled female cannot do so
- An Elizabethan collar inhibits cannibalism in queens
- The bitch should be attended at the birth of the litter (known as “parturition”) and puppies should be removed temporarily, if she is biting the puppies themselves in addition to the umbilical cord
- Bitches and queens with poor maternal behavior may exhibit the same behavior with subsequent litters

Excessive Maternal Behavior

- Cats that have stolen kittens should be separated from the natural mother and kittens
- Mothered objects (such as stuffed toys) should be removed from the “pseudopregnant” bitch (that is, the bitch having a false pregnancy)
- Food intake should be restricted to inhibit milk production (lactation)

Maternal Aggression

- The best treatment for excessive maternal aggression is to separate the kittens; weaning alone may not suffice because the presence of the kittens alone may sustain or even reinstate maternal aggression in a queen separated from her kittens for several weeks

PEDIATRIC BEHAVIOR PROBLEMS—CATS



BASICS

OVERVIEW

- Undesirable behaviors exhibited by kittens between birth and puberty—behaviors in this age range are particularly vulnerable to developmental and environmental influences
- Behaviors acquired during this period may be difficult to change; preventive measures are extremely important

GENETICS

- Possible influences for fearfulness in kittens by the tomcat (that is, the father cat)

SIGNALMENT/DESCRIPTION of ANIMAL

Species:

- Cats

Breed Predilections:

- None

Mean Age and Range:

- Precise data unknown

Predominant Sex:

- None

SIGNS/OBSERVED CHANGES in the ANIMAL

- Most common behavior problems of kittens are related to play, fearfulness, defensive aggression, and elimination
- Play is composed of components of other behaviors, often hunting or predatory behavior and fighting between cats; play can be solitary, with objects, or social (that is, with other kittens or cats)
- Social play often is accompanied by signals that indicate the activity is play and not a “serious” encounter
- During normal play, bites are inhibited and claws not extended fully; play is adjusted to the partner—if one partner escalates the intensity, the other usually follows suit
- If play gets too rough, one partner may signal that the activity is too rough (such as vocalize, quit, or become defensively aggressive and inflict injury)
- Play directed towards people or other animals in the house may be unwelcome, either due to frequency or intensity
- Fear and defensive behaviors include hiding, fleeing, and aggression
- Fear/defensive aggression often is characterized by flattened ears, hissing, and dilated pupils

Fear and Defensive Behaviors Due to Lack of Early Experience

- Behaviors associated with fear (such as dilated pupils, hair standing up, especially over the back and tail [known as “piloerection”], defensive postures, hissing, hiding, fleeing, aggression)
- Has always been afraid of people

Aggressive Play Directed Towards People

- Unsolicited attacks by kitten directed towards people
- Inhibited bites may indent the skin, and light scratches with claws—if person’s skin is soft or fragile, the wounds may break the skin
- If a person runs away, puts feet up, or tries to brush kitten away, the intensity of the play may increase—ambushes are common
- No vocalizations
- Generally starts when a kitten, but may continue into adulthood
- Usually seen in a single-kitten household
- Often ritualized, occurring in same locations and same time of day
- Often directed to specific persons

Uninhibited Aggressive Play Directed Towards People

- Signs similar to normal aggressive play, except more intense
- Bites are not as inhibited and usually break the person’s skin

Aggressive Play Directed Towards Other Cats in the Household

- Unsolicited attacks by kitten directed towards other cat
- Other cat, usually elderly, either runs and hides or responds by hissing, threatening, or seriously retaliating and attacking kitten

Normal Play Directed Towards Objects in the Household

- Bursts of solitary play that include intense running across household furnishings
- Shredding objects or propelling self along back underneath furniture
- Knocks over objects and removes them from horizontal surfaces

CAUSES

Fear and Defensive Behaviors Due to Lack of Early Experience

- No exposure to people when the kitten is between 3 and 7 weeks of age

Aggressive Play Directed Towards People or Other Cats in the Household

- Normal cat or species-typical behavior
- Lack of other outlets for play

Uninhibited Aggressive Play Directed Towards People

- Orphan-reared kitten with no littermates or other cats with which to play
- Rough play encouraged by people
- Teasing kitten

Normal Play Directed Towards Objects in the Household

- Normal cat or species-typical behavior

Fear and Defensive Behaviors Related to Early Trauma

- Normal behavior until kitten experienced traumatic event (such as abuse, attack by another animal)

Fear and Defensive Behaviors Related to Correction Techniques

- Normal behavior until kitten "corrected" by person (such as spanked, hit on the nose, yelled at, or chased)

RISK FACTORS

Aggressive Play Directed Towards People or Other Cats in the Household

- The only young cat in the household
- No appropriate outlets provided for normal play and exploration

Uninhibited Aggressive Play Directed Towards People

- The longer the delay between 3 weeks of age and when an orphan-reared kitten experiences play with other kittens and/or cats, the more likely uninhibited aggressive play will occur
- Adolescent or juvenile male human in household

Normal Play Directed Towards Objects in the Household

- Lack of environmental stimuli
- No appropriate toys available
- Little or no interactive play with people or other animals
- Only kitten or pet in household



TREATMENT

HEALTH CARE

- Outpatient

ACTIVITY

- Many pediatric behavior problems can be alleviated or reduced by enriching the kitten's environment (such as providing movable toys; engaging in interactive play; allowing the kitten access to windows; not shutting kitten in small, barren rooms)

DIET

- Undoubtedly influences development of nervous system and behavior, but specifics unclear
- Premium prenatal diet for the mother cat (queen) and premium kitten diets



MEDICATIONS

- None needed, unless fear and anxiety is extreme



FOLLOW-UP CARE

PATIENT MONITORING

- Two, 12 and 26 weeks after the initial consultation, re-check by phone or during subsequent visits

PREVENTIONS AND AVOIDANCE

- Kitten behavior problems can be prevented
- Kittens should experience positive interactions with people between 3 and 7 weeks of age
- Clients with children in the household specifically should be advised to prohibit roughhouse play with kittens
- Punishment may result in fear, anxiety, and defensive aggression in the kitten; avoid punishment of the kitten
- Behavior education (including advice from the veterinarian, pamphlets, videos, or books) at routine office visits or special kitten appointments

POSSIBLE COMPLICATIONS

- Defensive or aggressive behavior as an adult cat
- Injury to other animals or people
- A weakened bond with the pet and possible relinquishment to a shelter

EXPECTED COURSE AND PROGNOSIS

Normal Play Behaviors Directed Towards People, Other Cats, and Household Objects

- Appropriately followed treatment protocols should result in quick reduction or resolution of problem; if behavior not resolving, follow-up appointment is needed

Uninhibited Aggressive Play Directed Towards People

- Guarded prognosis

Fear and Defensive Behaviors Due to Lack of Early Experience or Related to Early Trauma

- It may take months, or even years, to acclimate the kitten to people; kittens will vary in the degree to which they acclimate; some kittens may never be comfortable around people
- If clients report no improvement, it may be that they are inadvertently reinforcing escape and defense behaviors by advancing towards the kitten
- The longer the interval between 3 weeks of age and lack of exposure to people, the poorer the prognosis
- The more intense the early trauma, the poorer the prognosis

Fear and Defensive Behaviors Related to Correction Techniques

- Should resolve within weeks, if clients follow advice
- The more frequent the correction, the poorer the prognosis



KEY POINTS

Aggressive Play Directed Towards People

- The most effective treatment is to acquire an additional kitten of the same size and temperament; the kittens will play with each other, and attacks directed towards people should diminish
- Interactive play with kitten using toys or objects that move; should play on a regular, daily basis
- Identify circumstances in which the attacks occur and redirect the play to another object (such as a string, ball)
- Do not encourage escalation of play with evasive actions or mild aversive techniques
- Can use a startling stimulus (such as water, foghorns, compressed air, and citronella spray) as a punisher—such a stimulus will not work

unless it is used every time the kitten attacks

- Do not hit, kick, or snap kitten on nose with fingers—such actions frequently elicit an immediate serious aggressive response from kitten and/or induce residual fear and fear-induced aggression towards that person
- Frequent trimming of tips of claws helps reduce damage

Aggressive Play Directed Towards Other Cats in the Household

- Acquire an additional kitten of the same size and temperament of the problem kitten
- If acquiring another kitten is not an option, the problem kitten and older cat must have restricted access to each other
- Startling, punitive techniques would affect the older cat aversively
- Interactive play with kitten using toys or objects that move on a regular, daily basis

Uninhibited Aggressive Play Directed Towards People

- Treatments would be similar to those used for normal aggressive play, except it is unwise to acquire a second kitten as the problem kitten might injure a second kitten
- Declawing is an option although controversy exists about the humaneness of this procedure, several studies indicate that declawing is not psychologically harmful to cats—declawing may be preferable to relinquishment of the kitten to animal control or an animal shelter

Normal Play Directed Towards Objects in the Household

- Put valuable, breakable, or dangerous objects away
- Provide appropriate toys for kitten
- Interactive play with kitten using toys or objects that move on a regular and daily basis
- Prohibit access to items
- “Booby-traps” or self-activated punishers might be used to keep kitten away from a few select objects or areas
- Provide scratching posts
- Frequent trimming of tips of claws or Soft Claws® or beads applied to claws
- Declawing is an option; although controversy exists about the humaneness of this procedure, several studies indicate that declawing is not psychologically harmful to cats—declawing may be preferable to relinquishment of the kitten to animal control or an animal shelter

Fear and Defensive Behaviors Due to Lack of Early Experience

- Gradual exposure to people without forcing any interactions
- In general, the kitten should be housed where it is comfortable, can remove itself from view but be continuously/very frequently aware of people
- Generally counterconditioning is required—initially, food can be put in or near the hiding area; gradually the food is placed further from the hiding area and closer to where a person is stationary; no attempt should be made to grab the kitten; the food can be left progressively further from the hiding place while people engage in their normal activities and the food eventually may be placed on a person’s lap
- Toys on strings can be used to entice the kitten to play; eventually the kitten may accept stroking, then holding
- Important principles to remember are to let the kitten make the advances—not the person—and avoid scaring the kitten; frightened kittens can bite and scratch

Fear and Defensive Behaviors Related to Early Trauma

- Identify the stimuli that elicit the fearful and/or defensive behaviors
- Employ behavior modification techniques, as described previously

Fear and Defensive Behaviors Related to Correction Techniques

- Identify and cease inappropriate punitive behaviors of people
- Identify the stimuli that elicit the fearful and/or defensive behaviors
- Employ behavior modification techniques, as described previously

PEDIATRIC BEHAVIOR PROBLEMS—DOGS



BASICS

OVERVIEW

- For the most part, these problems include behaviors that are normal and common to most puppies, but they are not acceptable to the family
- The undesirable behaviors require some degree of modification and “shaping” to become acceptable; “shaping” is a behavioral technique that gradually directs the puppy to the desired behavior—the puppy is rewarded for a response that is similar to the desired behavior in a stepwise fashion, until the desired behavior is accomplished
- Training problems include destructive chewing, playbiting, jumping on people, and getting on counters or furniture

GENETICS

- Activity levels and behaviors of young puppies are likely to be similar to those of their parents
- Some problem behaviors may be more common in certain breeds (such as unruly, activity-related problems in working-breed dogs and digging by terriers)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilection

- Working breeds selected for high energy levels

Mean Age and Range

- Four- to nine-months of age, but may persist until late in the second year

Predominant Sex

- Somewhat increased frequency and intensity in male dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

Destructive Chewing

- The pet chews and damages family members’ furniture and possessions; initially occurs in the presence of family members, but may become limited to owner-absent periods once the pet has been caught and punished several times

Playbiting

- The pet bites hands, legs, and/or clothing; bites usually are inhibited, but can cause injuries owing to sharp deciduous (“baby”) teeth
- Growling and barking may be present, but usually has a tone with a higher pitch than that associated with more serious types of aggression (such as fear or possessive aggression)
- Play attacks usually are triggered by some movement by a family member, but can be very spontaneous without apparent provocation or stimulus

Jumping on People

- The pet jumps up against and places paws against family members and/or visitors; typically occurs during greetings and when the pet is excited, but may occur when the pet wants attention or something the person is holding

Getting On Counters/Furniture

- The pet gets on furniture and counters to access objects to chew or eat
- The pet also may jump on furniture during play, to get attention, or to rest

CAUSES

General

- Inadequate owner control, supervision, training, exercise, and/or mental stimulation can be underlying causes of these behavior problems

Destructive Chewing

- Poor nutrition, inadequate amounts of food
- Mice or other small mammals in the walls or flooring
- Food spilled on carpeting or furniture
- Exploratory behavior; insufficient or uninteresting toys

- Escape behavior

Playbiting

- Rough play, teasing, and encouraging the pet to bite hands and feet

Jumping on People

- Long confinement periods, especially in a very small enclosure
- Excited greetings by family members and visitors
- Rough play

Getting on Counters/Furniture

- Insufficient or uninteresting toys
- Tempting objects or food left on furniture
- No comfortable surface on the floor on which to rest



TREATMENT

HEALTH CARE

- Outpatient

ACTIVITY

- Provide as much vigorous exercise as possible that is within acceptable health parameters for the individual puppy
- “Fetch/Drop It” is an excellent game for providing exercise and reminding the pet that the owner has control of resources (for example, toys and food); it also will help family members retrieve objects from the pet that he shouldn’t have—using two objects, throwing one and holding the other to throw once the pet returns with the first object can help keep the game going in puppies that may not drop the toy

DIET

- Feed enough food at optimum times to keep the pet satisfied, in order to decrease its motivation to get on counters, get into trash, guard food or chew on inanimate objects
- Your pet’s veterinarian will make recommendations on diet and amount to feed; food requirements can vary considerably from puppy to puppy



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drugs generally are not indicated
- On rare occasions, a small dose of a phenothiazine tranquilizer (such as acepromazine) or an antihistamine (such as diphenhydramine) might be considered for sedation during the early training period when friends visit and the pet is not yet under control



FOLLOW-UP CARE

PATIENT MONITORING

- Follow-up appointments must be determined on a case-by-case consideration
- Phone call follow-ups at approximately 10 days, 20 days, and six weeks following the initial visit are usually helpful
- A trained veterinary support staff member can play an important roll in helping with follow-up calls

PREVENTIONS AND AVOIDANCE

- Provide an adequate amount of supervision and confinement
- Begin food-lure-reward obedience training in the home at seven to eight weeks of age; enroll in a puppy class at eight to ten weeks of

age

- Large amounts of physical exercise and mental stimulation
- The veterinary staff will provide information about normal young pet behavior and needs (especially mental and physical stimulation) during various growth phases, so the family knows what to expect
- Safe and interesting toys

POSSIBLE COMPLICATIONS

- Damaged household objects and clothing
- The family's food eaten by the pet
- Intestinal foreign bodies and blockages or obstructions
- Minor skin injuries to the person from playbites
- A guest is knocked down and injured
- A weakened bond with the pet and possible relinquishment to an animal shelter

EXPECTED COURSE AND PROGNOSIS

- Prognosis is generally good; the frequency and intensity of the behaviors will decrease with age
- Jumping up on people and playbiting usually can be controlled quickly, if the family is consistent with training
- The tendency to chew occasionally on the family's possessions or explore counters for food and other objects may last until 12 to 24 months of age, when the pet becomes behaviorally mature and less active



KEY POINTS

General

- Use of rewards and punishment, including timing, consistency, value and intensity; harsh or physical punishment should be avoided—never strike the pet, thump its nose, shake it by the scruff, roll it on its back, or squeeze the lips against its teeth in an attempt to stop mouthing or biting—these approaches may increase the severity of the problem, ruin the bond with the pet and lead to more serious problems, such as fear and aggression
- Family members should look constantly for and reward acceptable behaviors
- Teach the pet to sit on command by using food-lure training

Destructive Chewing

- Provide interesting toys; experiment with different types of toys to find types the pet prefers
- Offer toys in which small amounts of food can be wedged or hidden to make them more attractive
- Reward acceptable chewing with praise and by tossing treats when the pet chews its toys
- Keep forbidden objects out of reach
- Close doors and use baby gates to restrict access to objects of interest to the puppy
- Spray objects that need to be protected with safe, aversive-tasting substances (prior to use, spray a small area as a test to ensure that spray will not damage the object [for example, to check fabric for colorfastness])
- Use a motion-activated alarm to keep the pet away from objects that need to be protected
- Interrupt any unacceptable chewing with a sharp “No,” the noise of a shake can (a can with a few pennies in it that makes noise when shaken), the “hiss” from a can of compressed air, or an air horn—any of these interruptive methods should be used with some attention to the pet's temperament; they should be minimal in intensity so that a fear response is not elicited from the pet
- Close supervision or safe confinement of the pet may be necessary for up to two years of age

Playbiting

- Provide plenty of exercise to reduce reactivity and impulsivity
- Have toys available at all times to toss and distract the pet; use toys in which small amounts of food can be wedged or hidden to divert the pet's attention and keep it occupied
- Use a leash and head halter, as needed for more control
- Place the pet in time-out when it is out of control and the family cannot devote the time needed to “shape” the behavior or wear the pet out with exercise
- Avoid games that encourage playbiting hands or feet
- The puppy should be enrolled in puppy classes as early as possible (eight to ten weeks of age)
- Take control of the pet by controlling resources (for example, toys and food) and making it sit before receiving toys, food, play, and attention

- Ignore any pushy social behavior by the puppy (such as whining, barking, or pawing for attention)
- Saying “Ouch” very loudly and walking away from the pet should immediately interrupt any hard bites during play
- Physical corrections should be avoided, because they can cause fear, anxiety, and aggression

Jumping on People

- Avoid play and games during which the pet jumps up on people
- Teach the puppy to sit on command
- Every time the puppy approaches a person for attention or to greet someone, quickly place a small treat or toy in front of its mouth and ask it to sit
- If the puppy jumps up, the behavior can be interrupted with a sharp noise (as previously described in ***Destructive Chewing***) or a head halter can be used to increase control and prevent jumping
- All family members must be very consistent in responding to this problem and shaping the pet’s behavior

Getting on Counters/Furniture

- Keep food and interesting objects off counters and furniture during the early training period
- Constantly supervise the puppy or place the pet in a safe confinement area
- Provide interesting toys for mental stimulation and to keep the puppy focused on objects on the floor
- Use motion-activated alarms or air canisters to teach the puppy to stay off furniture and counters when unsupervised
- Keep the puppy well fed, so it is not hungry and, therefore, less likely to look for food on tables and counters
- Provide a doggie bed on the floor

EXTRAHEPATIC BILE DUCT OBSTRUCTION (BLOCKAGE OF THE EXTRAHEPATIC OR COMMON BILE DUCT)



BASICS

OVERVIEW

- The liver is the largest gland in the body; it has many functions, including production of bile (a fluid substance involved in the digestion of fats); bile ducts begin within the liver itself as tiny channels to transport bile—the ducts join together to form larger bile ducts and finally enter the extrahepatic or common bile duct, which empties into the upper small intestine; the system of bile ducts is known as the “biliary tree”
- The gallbladder is the storage unit for bile; bile is stored until it is needed for fat digestion
- “Extrahepatic bile duct obstruction” is a blockage of the biliary tree at the level of the extrahepatic or common bile duct or at the level of the liver bile ducts (may involve one, several, or all ducts, depending on the disorder) that results in the flow of bile being decreased or stopped (known as “cholestasis”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predisposition

- Animals with increased likelihood of developing inflammation of the pancreas (known as “pancreatitis”)—breeds having high levels of lipids (compounds that contain fats or oils) in their blood (known as “hyperlipidemic breeds”), such as miniature schnauzers, Shetland sheepdogs

Mean Age and Range

- Middle-aged to old animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on underlying disorder
- Progressive sluggishness (lethargy)
- Intermittent illness
- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)
- Pale or grayish coloration to the stools (known as “acholic feces”), due to the lack of bile pigments that cause the normal brown color of bowel movements: indicate complete blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction)
- Increased appetite (known as “polyphagia”)—complete blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction) causes poor digestion of fats due to lack of bile flow
- Bleeding tendencies within 10 days of obstruction
- Weight loss
- Enlarged liver (known as “hepatomegaly”)
- Orange urine

CAUSES

- Associated with diverse disorders
- Presence of hard, solid material in the bile duct or gall bladder (known as “cholelithiasis”)
- Inflammation of the common bile duct (known as “choledochitis”); the extrahepatic or common bile duct empties into the upper small intestine
- Cancer
- Malformation of bile ducts
- Parasitic infestation (flukes in cats)
- Compression of the bile duct from surrounding tissues (such as lymph nodes, cancer, inflammation of the pancreas [pancreatitis], diaphragmatic hernia)
- Scarring of the bile duct (known as “duct fibrosis”), such as secondary to trauma, inflammation of the lining of the abdomen (known as “peritonitis”), inflammation of the pancreas (pancreatitis); major duct involvement in some cats with inflammation of the bile duct or biliary tree (known as “cholangitis”) and inflammation of the bile ducts and liver (known as “cholangiohepatitis”)
- Narrowing of the bile duct, secondary to blunt trauma, surgical manipulations/procedures



TREATMENT

HEALTH CARE

- Inpatient—surgical treatment of blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction)
- Fluid therapy—depends on underlying conditions; rehydrate and provide maintenance fluids before general anesthesia and surgery
- Water-soluble vitamins—B complex in intravenous fluids

ACTIVITY

- Dependent on patient status, and if patient has blood-clotting disorder secondary to liver disease

DIET

- Maintain nitrogen balance: avoid protein restriction
- Restrict fat—abnormal fat digestion caused by lack of intestinal bile acids (used in normal digestion of fats)
- Supplement fat-soluble vitamins, especially vitamins E and K; administer vitamins by injection

SURGERY

- Surgical exploration—imperative for treating and determining underlying cause
- Surgical treatment of blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction)
- Remove masses; remove gallstones (known as “choleliths”) and thickened bile
- May need to remove the gallbladder in some cases
- May need to produce a new connection between the biliary tree and the small intestines (known as a “biliary-enteric anastomosis” if the bile duct obstruction cannot be resolved or if the patient has scarring inflammation of the pancreas (known as “fibrosing pancreatitis”) or cancer
- Low blood pressure (known as “hypotension”) and slow heart rate (known as “bradycardia”)—may occur with biliary tree manipulation during surgery
- Surgical biopsies/samples—submit tissues and bile samples for bacterial cultures; submit tissues for microscopic examination to determine type of tissue (such as inflammation or cancer); inspect samples for evidence of bacterial infection and presence of parasite (flake) eggs
- Sclerosing inflammation of the bile duct or biliary tree (cholangitis) in cats (characterized by thickening or hardening of the biliary and/or liver tissues)—clinically may mimic blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction) since disease may involve extrahepatic biliary structures; will not respond to biliary tree decompression; liver biopsy essential for diagnosis



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Vitamin K1—necessary for normal blood clotting; administer 12 to 36 hours before surgery; treat early to allow response before surgical manipulations
- *Before surgery*—broad-spectrum antibiotics for potential biliary infections, as surgical manipulations may lead to spread of bacteria into the blood stream (known as “bacteremia”)
- Antioxidants—vitamin E (tocopherol); *S*-adenosylmethionine (SAME, Denosyl® SD4)
- Ursodeoxycholic acid—to improve secretion of bile (ensure adequate hydration); should be used after the bile-duct obstruction has been relieved
- Agents that reduce stomach acid and protect the stomach—famotidine (H₂-blocker) or omeprazol (pump inhibitor) combined with sucralfate, if medications administered by mouth are tolerated; stagger sucralfate administration from other oral medications to avoid drug interactions



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on underlying conditions
- Monitor blood work (serum chemistry profile, especially total bilirubin values [reflect effectiveness of relief of bile-duct obstruction—should decline to near normal within days] and liver enzymes [decline slowly])
- Complete blood count (CBC)—repeat every two to three days initially, if patient has generalized bacterial infection (known as “sepsis”)
- Inflammation of the lining of the abdomen due to bile leakage (known as “bile peritonitis”)—evaluate fluid accumulation in the abdomen (such as by feeling the abdomen [known as “palpation”], ultrasound examination [preferred], tapping the abdomen to withdraw accumulated fluid [known as “abdominocentesis”])
- Determine necessity for pancreatic enzyme supplementation based on site of the new connection between the biliary tree and the small intestines (biliary-enteric anastomosis); pancreatic enzymes are digestive enzymes that breakdown dietary proteins, fats, and starches in the intestines

POSSIBLE COMPLICATIONS

- Inflammation of the lining of the abdomen due to bile leakage (bile peritonitis)
- Repeated narrowing or stricture of the bile duct
- Narrowing or stricture of the new connection between the biliary tree and the small intestines (biliary-enteric anastomosis)
- Severe intestinal bleeding—high blood pressure in the intestinal blood vessels (known as “hypertensive enteric vasculopathy”) with blood-clotting disorder due to vitamin K deficiency
- Bleeding during surgery
- Low blood pressure (hypotension) and slow heart rate (bradycardia)—may occur with biliary tree manipulation during surgery

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying disease
- Prognosis good if inflammation of the pancreas (pancreatitis) resolves; bile-duct patency may return
- Permanent scarring of the liver tissue surrounding the biliary tree from blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction)
- Sclerosing inflammation of the bile duct or biliary tree (cholangitis) in cats (characterized by thickening or hardening of the biliary and/or liver tissues)—clinically may mimic blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction) since disease may involve extrahepatic biliary structures; will not respond to biliary tree decompression; liver biopsy essential for diagnosis



KEY POINTS

- Surgical treatment to relieve bile-duct obstruction is essential; obstruction will lead to progressive damage and scarring of the biliary tree and liver (known as “biliary cirrhosis”) within 6 weeks; exception is inflammation of the pancreas (pancreatitis) causing blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction) that may self-resolve within 2 to 3 weeks
- Surgical success is based on underlying cause, results of liver biopsy, and specimen cultures

DISEASE CAUSED BY *BLASTOMYCES*, A TYPE OF FUNGUS (BLASTOMYCOSIS)



BASICS

OVERVIEW

- A generalized (systemic), fungal infection caused by the soil organism, *Blastomyces dermatitidis*

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Occasionally cats

Breed Predislection

- Large-breed dogs weighing 55 lbs (25 kg) or more, especially sporting breeds; may reflect increased exposure rather than increased likelihood of developing disease

Mean Age and Range

- Dogs—most common 2 to 4 years of age; uncommon after 7 years of age
- Cats—young to middle-aged

Predominant Sex

- Dogs—males in most studies

SIGNS/OBSERVED CHANGES in the ANIMAL

- Weight loss
- Decreased appetite
- Cough and difficulty breathing (known as “dyspnea”)
- Eye inflammation and discharge
- Lameness
- Draining skin lesions

Dogs

- Fever up to 104.0° F (40° C)—approximately 50% of patients
- Harsh, dry lung sounds associated with increased breathing effort—common
- Generalized or regional enlarged lymph nodes (known as “lymphadenopathy”), with or without skin lesions
- Inflammation of the iris (the colored part of the eye) and other areas in the front part of the eye (known as “uveitis”), with or without secondary increased pressure within the eye (known as “secondary glaucoma”) and inflammation of moist tissue of the eye (known as “conjunctivitis”), discharge from the eye, and fluid build-up in the clear part of the eye (known as “corneal edema”)
- Lameness—common because of fungal infection/inflammation of the bone marrow and bone (known as “osteomyelitis”)
- Enlargement of the testicles and prostate—occasionally seen

Cats

- Increased breathing effort
- Nodular (known as “granulomatous”) skin lesions

CAUSES

- Inhaling fungal spores of *Blastomyces dermatitidis*

RISK FACTORS

- Depend on environmental and soil conditions that favor growth of *Blastomyces*
- Wet environment—fosters growth of the fungus; banks of rivers, streams, and lakes or in swamps; most affected dogs live within 400 m of water
- Exposure to recently excavated areas
- Most common along the Mississippi, Ohio, and Tennessee River basins; also found in the area of the Great Lakes, the St. Lawrence River, and has been found in Colorado



TREATMENT

HEALTH CARE

- Usually outpatient with antifungal treatment, using itraconazole administered by mouth

- Dogs with severe difficulty breathing (dyspnea)—require an oxygen cage for a minimum of 1 week before lung improvement is sufficient for comfort in room air; many have worsening of their lung disease during the first few days of treatment, owing to an increase in the inflammatory response after the *Blastomyces* organisms die

ACTIVITY

- Patients with breathing compromise must be restricted

DIET

- Palatable and high-quality to stimulate the appetite

SURGERY

- Removal of an abscessed lung lobe may be required when medical treatment cannot resolve the infection
- Blind eyes should be removed surgically (known as being “enucleated”) to eliminate potential sites of residual infection



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Itraconazole (Antifungal Medication)

- Dogs—as directed by your pet’s veterinarian; administer by mouth with a fat-rich meal, such as canned dog food, to increase absorption of the drug into the body
- Cats—open the 100-mg capsules containing pellets and mix the desired amount of pellets with palatable food, as directed by your pet’s veterinarian
- Treat for a minimum of 60 days or for 1 month after all signs of disease have disappeared
- Beware of generic itraconazole, as drug absorption is not reliable

Other Medications

- Dogs with nervous system signs should be treated with amphotericin B
- Ketoconazole—cheaper alternative to itraconazole; lower response rate and higher recurrence rate



FOLLOW-UP CARE

PATIENT MONITORING

- Blood work (serum chemistry)—monthly to monitor for liver toxicity or if lack of appetite (known as “anorexia”) develops
- Chest X-rays—determine duration of and response to treatment; considerable permanent changes in the lungs may occur after the infection has resolved, making determination of persistent active disease difficult

PREVENTIONS AND AVOIDANCE

- Location of environmental growth of *Blastomyces* organisms unknown; thus difficult to avoid exposure; restricting exposure to lakes and streams could be done, but is not very practical
- Dogs that recover from the infection are probably immune to reinfection

POSSIBLE COMPLICATIONS

- Blindness
- Death

EXPECTED COURSE AND PROGNOSIS

- Death—25% of dogs die during the first week of treatment; early diagnosis improves chance of survival
- Severity of lung involvement and invasion into the brain affect prognosis
- Recurrence—about 20% of dogs; usually within 3 to 6 months after completion of treatment, even with 60 to 90 days of treatment; may occur up to 15 months after treatment; a second course of itraconazole treatment will cure most patients; drug resistance to itraconazole has not been observed
- With early detection of blastomycosis, the prognosis in cats appears similar to dogs



KEY POINTS

- Treatment is costly and requires a minimum of 60 to 90 days

- Considerable permanent changes in the lungs may occur after the infection has resolved, making determination of persistent active disease difficult
- Not spread from animals to people, except through bite wounds; inoculation of *Blastomyces* organisms from dog bites has been reported
- Blastomycosis is acquired from an environmental source; people in the family may have been exposed at the same time as the pet; common source exposure has been documented in duck and coon hunters
- Family members with breathing problems and skin lesions should inform their physicians that they may have been exposed to *Blastomyces*

INFLAMMATION OF THE EYELIDS (BLEPHARITIS)



BASICS

OVERVIEW

- Inflammation of the outer (skin) and middle portion (muscle, connective tissue, and glands) of the eyelid, usually with secondary inflammation of the moist tissue lining the inner surface of the eyelid (known as the “palpebral conjunctiva”)
- May be associated with bacterial infection, self-trauma to the eyelids (as from rubbing or scratching), or disorders of the meibomian glands
- The meibomian glands are located along the edge of the back part of the eyelids, just under the lining (palpebral conjunctiva) of the inner surface of the eyelid; they produce secretions along the edge of the eyelid to help hold tears within the eye

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Listed under causes

SIGNS/OBSERVED CHANGES in the ANIMAL

- Discharge from the eye(s); may be clear or may contain mucus and/or pus
- Squinting or spasmodic blinking (known as “blepharospasm”)
- Redness of the eyelid (known as “hyperemia”), fluid build-up in the eyelid (known as “edema”), and thickening of the eyelid
- Itchiness (known as “pruritus”)
- Scratching and/or rubbing the face and eyelids, leading to skin trauma, with or without bleeding and dried discharge (skin lesions are known as “excoriations”)
- Loss of pigment (known as “depigmentation”) of the skin and/or hair
- Loss of hair (known as “alopecia”)
- Swollen, cream-colored meibomian glands; the meibomian glands are located along the edge of the back part of the eyelids, just under the lining (palpebral conjunctiva) of the inner surface of the eyelid; they produce secretions along the edge of the eyelid to help hold tears within the eye
- Elevated, pinpoint openings of the meibomian glands
- Abscesses
- Scales and dried discharge on the surface of a skin lesion (known as “crusts”)
- Small, raised skin lesions (known as “papules”) or small, raised skin lesions containing pus (known as “pustules”)
- Single or multiple, small red swellings
- Coexistent inflammation of the moist tissues of the eye (known as “conjunctivitis”) and/or inflammation of the cornea (known as “keratitis”); the cornea is the clear part of the eye, located in the front of the eyeball

CAUSES

Congenital (present at birth) Disorders

- Eyelid abnormalities—may promote self-trauma or moist inflammation of the skin (known as “dermatitis”)
- Prominent folds of skin around the nose; abnormal eyelashes that turn inward, against the cornea (the clear part of the eye, located in the front of the eyeball; condition known as “trichiasis”); and lower lid “entropion,” in which the lower eyelid is curled inward toward the eyeball—shih tzus, Pekingese, English bulldogs, Lhasa apsos, pugs, Persian and Himalayan cats
- Two rows of eyelashes present in a single eyelid (known as “distichia”)—shih tzus, pugs, golden retrievers, Labrador retrievers, poodles, English bulldogs
- “Ectopic cilia,” in which one or more eyelashes grows in an unusual location (may grow through the conjunctiva, leading to irritation of the eye)
- Lateral lid “entropion,” in which the outer portion of the eyelid (away from the nose) is curled inward toward the eyeball—Chinese shar peis, chow chows, Labrador retrievers, rottweilers; adult cats (rare)
- Condition in which the eyelids do not close completely (known as “lagophthalmos”)—short-nosed, flat-faced (known as “brachycephalic”) breeds of dogs; Persian, Himalayan, and Burmese cats
- Deep pocket in the corner of the eye (closest to the nose)—dogs with long heads (known as “dolichocephalic dogs,” such as collies)
- Masses composed of “displaced” skin, frequently with long hair (masses known as “dermoids”)—rottweilers, dachshunds, and others; Burmese cats

Allergic Disorders

- “Allergy” is an unusual sensitivity to a substance (such as pollen)—the immune system responds to the presence of the substance leading to signs (such as itchiness); “antigen” is a substance (such as pollen) that induces a sensitivity or immune response; “antibody” is a protein that is produced by the immune system in response to a specific antigen—when the body is exposed to the antigen, the antibody responds, causing the signs of the allergic response
- Various allergic disorders can cause inflammation of the eyelids (blepharitis); they include atopy; food or medication allergies;

insect-bite allergy; flea-bite hypersensitivity; inhalant allergies; *Staphylococcus* hypersensitivity

Bacterial Infections

- Localized abscess of eyelid glands (known as a “hordeolum”)—usually staphylococcal infection; may be external (sty in young dogs) or internal (in old dogs, involves one or more meibomian glands); the meibomian glands are located along the edge of the back part of the eyelids, just under the lining (palpebral conjunctiva) of the inner surface of the eyelid; they produce secretions along the edge of the eyelid to help hold tears within the eye
- Generalized bacterial inflammation of the eyelids (blepharitis) and meibomian glands (known as “meibomianitis”)—usually caused by *Staphylococcus* or *Streptococcus*
- Nodular inflammatory lesions containing pus (known as “pyogranulomas”)
- Allergic reaction to skin bacteria (known as “*Staphylococcus* hypersensitivity”)—young and old dogs

Tumors or Cancer

- Benign tumors (known as “sebaceous adenomas”) and cancer (known as “sebaceous adenocarcinomas”)—originate from a meibomian gland; the meibomian glands are located along the edge of the back part of the eyelids, just under the lining (palpebral conjunctiva) of the inner surface of the eyelid; they produce secretions along the edge of the eyelid to help hold tears within the eye
- Squamous cell carcinoma—white cats
- Mast cell tumors—may masquerade as swollen, reddened lesion; mast cell tumors contain mast cells; mast cells are immune-system cells that frequently are located near blood vessels in the skin; mast cells contain histamine; they usually are involved in allergy and inflammation

Other Disorders

- External trauma—eyelid lacerations; thermal or chemical burns
- Fungal or mycotic infections
- Parasitic—demodectic mange (demodicosis); sarcoptic mange; *Cuterebra* or *Notoedres cati* infestation
- Inflammatory enlargement of a meibomian gland in the eyelid (known as “chalazia” [plural] or “chalazion” [singular])—sterile, yellow-white, painless meibomian gland swellings caused by a granulomatous inflammatory response to escape of meibum (the substance secreted by the glands) into surrounding eyelid tissue; the meibomian glands are located along the edge of the back part of the eyelids, just under the lining (palpebral conjunctiva) of the inner surface of the eyelid; they produce secretions along the edge of the eyelid to help hold tears within the eye
- Nutritional disorders—zinc-responsive skin abnormality (Siberian huskies, Alaskan malamutes, puppies); fatty acid deficiency
- Hormonal or endocrine disorders—inadequate production of thyroid hormone (known as “hypothyroidism”) in dogs; excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”) in dogs; skin lesions secondary to diabetes mellitus (sugar diabetes)
- Viral infection—long-term (chronic) inflammation of the eyelids (blepharitis) in cats secondary to feline herpes virus-1 (FHV-1)
- Irritant—reaction to medications applied directly to the eye (topical ocular drugs); nicotine smoke in environment
- Inherited inflammatory disorder that affects the skin and muscles of unknown cause (condition known as “idiopathic familial canine dermatomyositis”)—collies and Shetland sheepdogs
- Inflammation of the border between the cornea (the clear part of the eye, located in the front of the eyeball) and the sclera (the white part of the eye, composed of a tough covering that protects the eyeball) characterized by the presence of nodules (condition is known as “nodular granulomatous episclerokeratitis”)—also known as “fibrous histiocytoma” and “collie granuloma” in collies; may affect the eyelids, cornea, or conjunctiva
- Eosinophilic granuloma (a mass or nodular lesion containing a type of white blood cell, called an eosinophil)—cats; may affect eyelids, cornea, or conjunctiva
- Eyelid contact with discharge containing pus (“tear burn”)
- Inflammation of the moist tissues of the eyelids and eye (conjunctivitis)
- Inflammation of the cornea (keratitis)
- “Dry eye” (known as “keratoconjunctivitis sicca” or “KCS”), such as with inadequate tear production
- Inflammation of the nasolacrimal sac, part of the tear drainage system (condition known as “dacryocystitis”)
- Disease involving the bony cavity containing the eyeball (known as “orbital disease”)
- Radiation therapy
- Medication that irritates the eye when it comes into contact with the eye—any drug; often the antibiotic, neomycin
- Unknown cause (so called “idiopathic disease”)—particularly in cats with long-term (chronic) idiopathic inflammation of the moist tissues of the eyelids and eye (conjunctivitis)

RISK FACTORS

- Breed susceptibilities to eyelid abnormalities that are present at birth (congenital abnormalities), such as “entropion,” in which the lower eyelid is curled inward toward the eyeball or “ectropion,” in which the eyelid is turned outward
- Outdoor animals—trauma
- Inadequate production of thyroid hormone (hypothyroidism)—may promote long-term (chronic) bacterial disease in dogs
- Canine seborrhea, in which the dog has a skin disorder causing dry or oily scales—may promote long-term (chronic) generalized inflammation of the meibomian glands (meibomianitis); the meibomian glands are located along the edge of the back part of the eyelids, just under the lining (palpebral conjunctiva) of the inner surface of the eyelid; they produce secretions along the edge of the eyelid to help hold tears within the eye



TREATMENT

HEALTH CARE

- Secondary disease—treat primary, underlying disease
- Suspected self-trauma to the eyelids—apply an Elizabethan collar
- Medications applied directly to the skin of the eyelids and/or eyes (known as “topical medications”) include antibiotics such as gentamicin, neomycin, Terramycin® and antiviral medication (such as trifluridine solution)—may cause a drug reaction leading to inflammation of the eyelids and conjunctiva (known as “blepharoconjunctivitis” [rare]); discontinuing the medication may resolve condition
- Cleanse eyelids—to remove dried discharge (crusts); apply warm compresses for 5 to 15 minutes, 3 to 4 times daily, avoiding surfaces of the eyes; may use saline, lactated Ringer’s solution, or a commercial eye cleansing agent (such as Eye•Scrub®); must clip hair around the eyes short

DIET

- Dietary changes only necessary with food allergy–induced inflammation of the eyelids (blepharitis)

SURGERY

- Temporary sutures placed in the eyelids to evert “curled-in” eyelids—spastic entropion (in which the eyelid is curled inward toward the eyeball) secondary to squinting or spasmodic blinking (blepharospasm); or in puppies before permanent surgical correction of entropion
- Repair eyelid lacerations
- Surgical repair of eyelid abnormalities



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antibiotics

- Systemic (given by mouth [orally] or by injection)—generally required for effective treatment of bacterial eyelid infections; may try amoxicillin–clavulanic acid, or cephalexin
- Topical (applied to the eyelids and/or eyes)—may try neomycin, polymyxin B, and bacitracin combination or chloramphenicol—avoid neomycin, if it is suspected of being irritating for the particular patient

Congenital (present at birth) Disorders

- Topical (applied to the eyelids) antibiotic ointment—until surgery is performed to prevent frictional rubbing of eyelid hairs or eyelashes on the surface of the eye
- Saline, lactated Ringer’s solution, or eye irrigant—regularly flush the pocket in the corner of the eye, nearest the nose, to remove accumulated debris

Allergic Disorders

- Allergic reaction to the bacteria on the skin (*Staphylococcus* hypersensitivity) with inflammation of the eyelids (blepharitis)—systemic (given by mouth [orally] or by injection) broad-spectrum antibiotics and systemic steroids (such as prednisolone); many patients respond dramatically to systemic steroids alone; systemic cyclosporine (to decrease the immune response) if condition does not respond to treatment with steroids
- Affected meibomian glands—systemic antibiotics, such as oral tetracycline or doxycycline or cephalexin for at least 3 weeks; topical (applied to the eyelids) antibiotics, such as polymyxin B and neomycin with 0.1% dexamethasone or topical 0.02% tacrolimus ointment; tacrolimus is a medication that decreases the immune response (an “immunosuppressive drug”)
- Failure of treatment—may try injections (so called “allergy shots”) of *Staphylococcus aureus* bacterin (Staphage Lysate SPL®)

Bacterial Infections

- Antibiotics based on bacterial culture and sensitivity testing
- While waiting for results of bacterial culture and sensitivity testing—treat with topical (applied to the eyelids) polymyxin B and neomycin with 0.1% dexamethasone ointment; plus a systemic (given by mouth [orally] or by injection) broad-spectrum antibiotic

External Trauma

- Topical (applied to the eyelids and/or eyes) antibiotic ointment—for spastic entropion (in which the lower eyelid is curled inward toward the eyeball) secondary to squinting or spasmodic blinking (blepharospasm) and pain to reduce friction until entropion is relieved surgically
- Systemic (given by mouth [orally] or by injection) antibiotics

Fungal or Mycotic Infection

- *Microsporium canis* infection—treatment includes 2% miconazole cream, 1% clotrimazole cream, or diluted povidone-iodine solution

(1 part to 300 parts saline) applied topically for at least 6 weeks

Parasitic

- Demodectic mange (demodicosis)—localized disease, diluted amitraz (Mitaban®); fairly safe around the eyes
- *Notoedres* infestation—lime-sulfur dips

Unknown Cause (Idiopathic Disease)

- Signs often controlled with topical (applied to the eyelids and/or eyes) polymyxin B and neomycin with 0.1% dexamethasone; occasionally also may administer systemic (given by mouth [orally] or by injection) prednisolone and/or a systemic (given by mouth [orally] or by injection) antibiotic

Other

- Eyelid lesions associated with “juvenile cellulitis” or “puppy strangles,” a condition in which the puppy develops enlarged lymph nodes, facial swelling, hair loss, and small, raised skin lesions containing pus (known as “pustules”)—usually benefit from treatment of the generalized condition



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on cause and type of treatment
- Bacterial infection—systemic (given by mouth [orally] or by injection) and topical (applied to the eyelids and/or eyes) treatment for at least 3 weeks; should notice improvement within 10 days
- Most common causes of treatment failure—inadequate concentration of antibiotics; failure to correct one or more factors that increase the likelihood of development of inflammation of the eyelids (blepharitis); stopping medications too soon

PREVENTIONS AND AVOIDANCE

- Depend on cause
- Minimize stress for cats with inflammation of the moist tissues of the eye caused by feline herpesvirus (known as “herpetic conjunctivitis”)

POSSIBLE COMPLICATIONS

- Scarring that affects the eyelids and results in eyelid abnormalities (such as inability to close the eyelids [lagophthalmos])
- Spastic entropion (in which the eyelid is curled inward toward the eyeball) secondary to squinting or spasmodic blinking (blepharospasm) and pain
- Inability to open eyelids—secondary to matting of discharge and hair
- Tear-film deficiency as a result of loss of proper secretions from the meibomian glands; the meibomian glands are located along the edge of the back part of the eyelids, just under the lining (palpebral conjunctiva) of the inner surface of the eyelid; they produce secretions along the edge of the eyelid to help hold tears within the eye
- Recurrence of bacterial infection or feline herpes virus-1 (FHV-1) inflammation of the eyelids and conjunctiva (blepharoconjunctivitis)

EXPECTED COURSE AND PROGNOSIS

- Depends on cause



KEY POINTS

- Most patients cannot be cured, but the condition often can be controlled medically
- No cure exists for feline herpes virus-1 (FHV-1) infection; clinical signs often recur when the cat is stressed
- Minimize stress for patients with inflammation of the moist tissues of the eye caused by feline herpesvirus (herpetic conjunctivitis)
- Keep the Elizabethan collar on at all times, when self-trauma is suspected

BLIND QUIET EYE



BASICS

OVERVIEW

- Loss of vision in one or both eyes, without externally apparent signs of inflammation of the eye

GENETICS

- Many causes of blind, quiet eye (such as cataracts [opacities in the lens] and progressive deterioration of the back of the eye [the back of the eye is the “retina;” condition known as “progressive retinal atrophy”]) have a genetic basis; the “lens” is the normally clear structure directly behind the iris (colored part of the eye) that focuses light as it moves toward the back part of the eye (retina)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Many causes of blind, quiet eye (such as cataracts [opacities in the lens] and progressive deterioration of the back of the eye [retina; condition is progressive retinal atrophy]) are often breed-specific

Mean Age and Range

- Many causes of blind, quiet eye (such as cataracts [opacities in the lens] and progressive deterioration of the back of the eye [retina; condition is progressive retinal atrophy]) are often age-specific
- Sudden blindness due to “sudden acquired retinal degeneration syndrome” or “SARDS”—tends to occur in old dogs
- Underdevelopment of the optic nerve (the nerve that runs from the back of the eye to the brain; condition known as “optic nerve hypoplasia”)—congenital (present at birth)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary with underlying cause
- Bumping into objects
- Clumsy behavior
- Reluctance to move
- Impaired vision in dim light

CAUSES

- Cataracts (opacities in the lens)—entire lens must become opaque to produce complete blindness; incomplete opacification may reduce performance of visually demanding tasks; the “lens” is the normally clear structure directly behind the iris (colored part of the eye) that focuses light as it moves toward the back part of the eye (retina)
- Loss of focusing power of the lens—rarely completely blinding
- Retina (back of the eye)—sudden acquired retinal degeneration syndrome (SARDS); progressive deterioration of the back of the eye (progressive retinal atrophy); separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid;” condition known as “retinal detachment”); taurine deficiency that leads to retinal degeneration in cats (taurine is an amino acid [protein] that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the retina); enrofloxacin toxicity in cats (enrofloxacin is an antibiotic); ivermectin toxicity in dogs and cats (ivermectin is a medication that kills a variety of parasites)
- Optic nerve (the nerve that runs from the back of the eye to the brain)—inflammation of the optic nerve (known as “optic neuritis”); tumor or cancer involving the optic nerve or adjacent tissues; trauma; underdevelopment of the optic nerve (optic nerve hypoplasia); lead toxicity; excessive traction on the optic nerve during surgical removal of one eyeball (procedure known as “enucleation”), resulting in trauma to the optic nerve going to the opposite eye (especially in cats and short-nosed, flat-faced [known as “brachycephalic”] dogs)
- Central nervous system blindness (known as “amaurosis,” in which the cause of the blindness is a lesion outside of the eye itself, usually involving a lesion in the brain)

RISK FACTORS

- Poorly regulated diabetes mellitus (“sugar diabetes”)—cataracts (opacities in the lens)
- Related animals with genetic cataracts or progressive deterioration of the back of the eye (retina; condition is progressive retinal atrophy)
- Generalized (systemic) high blood pressure (known as “hypertension”)—separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)
- Decreased oxygen (known as “hypoxia”) to the tissues of the central nervous system—blindness may become apparent after excessively deep anesthesia or revival from cardiac arrest



TREATMENT

HEALTH CARE

- Varies with underlying cause
- Try to obtain a definitive diagnosis on an outpatient basis, before initiating treatment
- Consider referral to a veterinary eye doctor (ophthalmologist)
- Most causes are not fatal, but must perform a diagnostic workup to rule out potentially fatal diseases
- Sudden acquired retinal degeneration syndrome (SARDS); progressive deterioration of the back of the eye (progressive retinal atrophy); deterioration of the optic nerve (the nerve that runs from the back of the eye to the brain; condition known as “optic nerve atrophy”); and underdevelopment of the optic nerve (optic nerve hypoplasia)—no effective treatment

ACTIVITY

- Separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)—recommend severely restricted exercise, until the retina is firmly reattached

DIET

- Calorie-restricted diet—to prevent obesity; owing to reduced activity level
- Cats with nutritionally induced disease of the retina (known as a “retinopathy”)—ensure diet has adequate levels of taurine; taurine is an amino acid (protein) that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the retina

SURGERY

- Cataracts (opacities in the lens); lens that has moved out of its normal position in the eye (known as a “luxated lens”), and some forms of separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)—best treated surgically



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Depend on cause
- If infectious disease is unlikely, and the likely diagnosis is either sudden acquired retinal degeneration syndrome (SARDS) or inflammation of the optic nerve (the nerve that runs from the back of the eye to the brain) behind the eyeball (known as “retrobulbar optic neuritis”)—steroids (prednisolone) may be administered; also may administer chloramphenicol or other broad-spectrum antibiotic
- Flunixin meglumine (dogs)—may try a single dose in place of steroids, if infectious causes have not been ruled out
- Chemotherapeutic drug used to decrease the immune response (azathioprine)—may be used to treat immune-mediated separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment), if steroids are not effective



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat ophthalmic examinations—as required to ensure that inflammation of the eye is controlled, and, if possible, vision is maintained
- Recurrence of vision loss—common in inflammation of the optic nerve (optic neuritis); may occur weeks, months, or years after initial presentation
- If treatment includes azathioprine (a chemotherapeutic drug used to decrease the immune response)—perform blood work, including a complete blood count (CBC), platelet count, and liver enzymes every 1 to 2 weeks for the first 8 weeks, then periodically

PREVENTIONS AND AVOIDANCE

- Animals with progressive deterioration of the back of the eye (progressive retinal atrophy) or genetic cataracts (opacities in the lens) should not be bred and related animals should be examined by a veterinarian to evaluate the eyes

POSSIBLE COMPLICATIONS

- Vary with underlying cause

- Permanent vision loss
- Loss of the eye
- Long-term (chronic) inflammation and pain of the eye
- Obesity from inactivity or as a sequela of sudden acquired retinal degeneration syndrome (SARDS)
- Death

EXPECTED COURSE AND PROGNOSIS

- Vary with underlying cause



KEY POINTS

- Most causes of a blind quiet eye are not painful and blind animals can lead a relatively normal and functional life
- The environment should be examined for potential hazards to a blind animal
- Animals with progressive deterioration of the back of the eye (progressive retinal atrophy) or genetic cataracts (opacities in the lens) should not be bred and related animals should be examined by a veterinarian to evaluate the eyes

BRACHYCEPHALIC AIRWAY SYNDROME (UPPER AIRWAY PROBLEMS SEEN IN SHORT-NOSED BREEDS)



BASICS

OVERVIEW

- Partial upper airway obstruction in short-nosed, flat-faced (brachycephalic) breeds of dogs and cats caused by any combination of the following conditions: narrowed nostrils (known as “stenotic nares”); overly long soft palate; turning inside-out of a portion of the voice box or larynx (known as “everted laryngeal sacculles”), such that the space for air to pass through the larynx is decreased; and collapse of the voice box or larynx (known as “laryngeal collapse”)
- An abnormally small windpipe or trachea (known as “hypoplastic trachea”) often is present as well, and can worsen breathing difficulty (respiratory distress)

GENETICS

- No specific genes have been identified
- Short-nosed, flat-faced, broad (brachycephalic) head shape was initially an inherited defect in development of skull bones; has been perpetuated by selective breeding in certain breeds of dogs and cats

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Common syndrome in brachycephalic breeds of dogs
- Cats—less likely to be severe enough to require treatment

Breed Predispositions

- Dogs—English bulldogs (most common), pugs, Boston terriers, boxers, Pekingese, Cavalier King Charles spaniels, shih tzu, Chinese shar pei, and others
- Cats—Persians and Himalayans

Mean Age and Range

- Young adults, most diagnosed by 2 to 3 years of age
- If diagnosed later than 4 years of age, another disease or condition may be adding to upper airway obstruction leading to diagnosis; older dogs may have a worse outcome postoperatively, but most have some improvement

SIGNS/OBSERVED CHANGES in the ANIMAL

- Snoring; high-pitched, noisy breathing (stridor); noisy breathing when inhaling (stertorous breathing)
- Rapid breathing (tachypnea), frequent panting
- Coughing and gagging, or difficulty eating and swallowing
- Inability to perform physical activity and worsening of condition during warm and humid weather
- Occasionally, fainting (syncope) and episodes of collapse
- Narrowed nostrils (stenotic nares)
- Increased breathing (respiratory) effort—may see pulling back of the lips with each breath, open-mouth breathing or constant panting, increased breathing rate, turning of the elbows away from the body (abduction of forelimbs) in an effort to open up the chest, increased abdominal effort during breathing
- If in severe breathing distress, may have increased difficulty in breathing while lying flat (known as “orthopnea”), even to point of reluctance to lie down; and bluish discoloration of skin and moist tissues of body (known as “cyanosis”)
- Increased body temperature (hyperthermia) may be present
- May appear anxious and resent or resist restraint

CAUSES

Brachycephalic airway syndrome results from inherited defects or developmental/growth defects of the upper airway. These defects include the following:

- Elongated soft palate—reported in up to 100% of cases in dogs
- Narrowed nostrils (stenotic nares)—reported in about 50% of cases in dogs; most common defect in cats
- Voice box or laryngeal disease—everted laryngeal sacculles and/or laryngeal collapse reported in about 30% of cases
- Abnormally small windpipe (hypoplastic trachea)

RISK FACTORS

- Brachycephalic breed
- Obesity—worsens airway obstruction; associated with poorer outcome following surgical correction; may contribute to backward or reverse flow of stomach contents into the esophagus (known as “gastroesophageal reflux”) and development of aspiration pneumonia
- Abnormally small windpipe (hypoplastic trachea) can result in diminished ability to clear mucus from the airway (decreased mucociliary clearance), worsened aspiration pneumonia, and increased airway resistance
- Warm, humid weather—increased panting can lead to fluid build-up (edema) in the tissues of the airway, further narrowing the airway

opening, and leading to increased body temperature

- Exercise—dogs often are unable to exercise because of airway compromise and low levels of oxygen in their blood (hypoxia); forced exercise may lead to rapid development of overheating and collapse
- Excitement—can cause increased panting, resulting in fluid build-up (edema), increased airway obstruction, and increased body temperature
- Sedation may cause relaxation of the muscles of throat (pharynx) and soft palate, and may cause complete airway obstruction
- Sleep may cause relaxation of the muscles of throat (pharynx) and soft palate, leading to further airway obstruction
- Allergic reactions—acute allergic reactions causing build-up of fluid (edema) in the tissues of the airway may lead to airway obstruction
- Lung disease (such as pneumonia, pulmonary edema)—will cause additional breathing compromise
- Endocrine disease (such as low levels of thyroid [hypothyroidism] or high levels of steroid [hyperadrenocorticism or Cushing's disease])—could worsen weight gain and cause excessive panting



TREATMENT

HEALTH CARE

- No treatment necessary for patients without clinical signs
- Avoid risk factors
- Surgery recommended for patients with significant clinical signs
- Emergency presentation with animal in severe breathing distress requires rapid medical intervention
- Oxygen supplementation
- If patient has high body temperature (hyperthermia), should be cooled with cool water and by directing a fan to blow over the patient (increase convective heat loss); intravenous (IV) fluids should be administered, up to a shock rate if has an extremely elevated body temperature (greater than 106° F)
- If the airway is completely obstructed, the airway must be opened; this may be accomplished by passing an endotracheal tube through the mouth and into the windpipe (orotracheal intubation) and/or by a surgical incision into the windpipe (temporary tracheostomy)
- Dexamethasone can be administered to reduce inflammation
- Patients need 24-hour monitoring because of risk of acute airway obstruction and death
- Breathing rate and effort, heart rate, pulse quality, color of gums and moist tissues (mucous membrane color), time for pink color to return to gums after blanching them with one's finger (capillary refill time), temperature, and other physical parameters should be monitored
- Pulse oximetry and arterial blood gases to determine oxygen levels in the blood may be monitored, depending on severity of condition
- Intravenous fluids are administered at maintenance rate and handling and stress are minimized

ACTIVITY

- Usually self-limited by the animal
- Dogs should not be forced to exercise, especially in warm weather

DIET

- Weight loss is recommended for all overweight dogs and cats
- For obese, stable patients, weight loss is recommended prior to surgery

SURGERY

- Evaluation for elongated soft palate generally is performed under general anesthesia when the patient stable
- Surgical incision into the windpipe (temporary tracheostomy) can be performed to facilitate exposure or to treat airway obstruction
- Narrowed nostrils (stenotic nares) are corrected by surgically removing a wedge of the nasal tissue and closing the incision in such a manner to allow the nostril to be enlarged
- Elongated soft palate is treated by surgically removing a section of the soft palate using surgical scissors or carbon dioxide laser
- Everted laryngeal sacculs are treated by surgically trimming the tissue
- Permanent surgical opening into the windpipe or trachea (tracheostomy) may be necessary if severe laryngeal collapse is present



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Dexamethasone to reduce fluid build-up (edema) and inflammation
- Broad-spectrum antibiotics are indicated if aspiration pneumonia is present, until culture and sensitivity results are obtained
- Omeprazole and cisapride may be beneficial in dogs with vomiting or regurgitation



FOLLOW-UP CARE

PATIENT MONITORING

- Postoperatively, 24-hour monitoring to observe for airway swelling and obstruction, which may require surgical incision into the windpipe (temporary tracheostomy)
- Breathing rate, effort, heart rate, pulse quality, mucous membrane color, capillary refill time, temperature, and other physical parameters should be monitored
- Understand clinical signs of breathing problems

PREVENTIONS AND AVOIDANCE

- Selection by breeders for dogs without severe conformational changes—may be difficult because breed standards encourage these structural changes
- Avoid risk factors

POSSIBLE COMPLICATIONS

- Overheating and heat stroke
- Aspiration pneumonia
- Death in about 10% of patients as a result of airway disease
- Most common postoperative complication is airway swelling and obstruction within the first 24 hours, may necessitate surgical incision into the windpipe (temporary tracheostomy)
- Continued breathing difficulty after corrective surgery
- Excessive shortening of the soft palate resulting in aspiration of food contents into the nasal cavity due to inability to close off the area between the nose and throat (known as the “nasopharynx”) during swallowing

EXPECTED COURSE AND PROGNOSIS

- Prognosis is good for improvement in breathing (about 60-80% of cases have good to excellent results following corrective surgery) but airway is still far from normal
- Prognosis better for dogs other than English bulldogs and for dogs that have correction of stenotic nares and elongated soft palate during same surgery
- Without surgery, prognosis is poor due to continued progression of brachycephalic airway syndrome
- Lifelong avoidance of risk factors recommended to decrease chance of developing clinical signs or worsening of disease



KEY POINTS

- Avoidance of risk factors is critical
- Dogs with brachycephalic airway syndrome are at increased anesthetic risk, and an even higher risk occurs if they also have obesity, cardiac disease, or aspiration pneumonia
- Corrective surgery often improves the clinical signs, but does not result in a completely normal airway
- The American Kennel Club will not allow a dog that has had surgery for stenotic nares or elongated soft palate to compete in AKC-sanctioned dog shows

BRAIN INJURY



BASICS

OVERVIEW

- Primary brain injury—direct result of initial insult to the brain; cannot be altered
- Secondary brain injury—alteration of brain tissue (such as bleeding or swelling) that occurs after the primary injury; can be prevented or improved with optimal supportive care

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Decline in the level of consciousness—implies progression of secondary brain injury from bleeding in the skull (known as “intracranial bleeding”), lack of blood flow to the brain (known as “ischemia”), or fluid build-up within the brain (known as “cerebral edema”)
- Seizure activity
- Evidence of trauma (to the head or other parts of the body)
- Bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”)
- Bruising; bleeding into tissues of the eyes
- Blood from the ears or nose—trauma with bleeding into the skull (intracranial bleeding)
- Separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid;” condition known as “retinal detachment”)
- Heart or breathing abnormalities
- Abnormalities of the skull—skull fractures
- Constricted or dilated pupils—may involve one or both eyes; pupils may be uneven in size; response of the pupils to light may be altered
- Abnormalities of the nervous system; abnormalities can change over time

CAUSES

- Head trauma
- Prolonged low levels of oxygen (known as “hypoxia”) or decreased or lack of blood flow (ischemia) to tissues in the brain
- Severe low levels of glucose (sugar) in the blood (known as “severe hypoglycemia”)
- Prolonged seizures
- Severe increase in body temperature (known as “hyperthermia”) or decrease in body temperature (known as “hypothermia”)
- Changes in concentrations of chemicals in the blood (such as sodium or glucose)
- Prolonged shock
- Increased blood pressure (known as “hypertension”)
- Migration of parasites through brain tissue
- Infectious diseases
- Immune-mediated diseases
- Thiamine (a B vitamin) deficiency
- Poisons
- Brain tumor
- Stoppage of the heart (known as “cardiac arrest”)
- Severe heart failure
- Blood clots to the brain
- Blood-clotting disorders, leading to bleeding in the brain
- Prolonged breathing compromise

RISK FACTORS

- Free-roaming animal—trauma, exposure to poisons
- Coexisting heart and lung disease
- Uncontrolled high levels of glucose (sugar) in blood (known as “hyperglycemia”)



TREATMENT

HEALTH CARE

- Goals of therapy: maximize oxygen levels in the brain; support blood pressure and blood flow to the brain; decrease pressure within the skull (known as “intracranial pressure”); decrease metabolism of the brain
- Avoid cough or sneeze reflex during passage of an endotracheal tube into the windpipe or trachea (known as “tracheal intubation”) or oxygen supplementation into the nose—cough or sneeze reflex may elevate pressure within the skull (intracranial pressure)
- Do not block blood flow in the jugular veins
- Carefully administer fluids; excessive administration of fluids can contribute to fluid build-up in the brain (cerebral edema)
- If suspect bleeding, administer crystalloids alone (normal saline or isotonic balanced solution); “crystalloids” are fluids that contain electrolytes (chemical compounds, such as sodium, potassium, chloride) necessary for the body to function, crystalloids generally are similar to the fluid content (plasma) of the blood and move easily between the blood and body tissues, example is lactated Ringer’s solution
- If low blood pressure (hypotension) or significant inflammation of blood vessels (known as “vasculitis”) is present, a combination of crystalloids with large-molecular-weight colloids may be administered; colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are hetastarch and Oxyglobin®
- Avoid high blood pressure (hypertension)
- Keep head level with body or elevate to a 20° angle; head should never be lower than the body
- Maintain unobstructed airways; use suction and humidify air, if the animal is intubated
- Lubricate the eyes
- Position recumbent animal on its breastbone (sternum); if lying on its side, turn the patient every 2 hours to avoid lung congestion
- Meticulous nursing care prevents secondary complications of recumbency
- Prevent fecal/urine soiling
- Maintain body temperature at normal temperature or mildly decreased temperature (slight hypothermia); avoid increased body temperature (hyperthermia)
- Maintain hydration with balanced fluid solutions

ACTIVITY

- Restricted

DIET

- Initiate nutritional support to meet increased metabolic demands
- Tube-feeding may be required for early nutritional support

SURGERY

- Surgery may be necessary for depressed skull fracture, computed tomography (CT or CAT scan) or magnetic resonance imaging (MRI) evidence of surgical problem, or penetrating foreign body



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Elevated Intracranial Pressure

- Lower intracranial pressure by increased breathing rate to decrease carbon dioxide levels (known as “hyperventilation”), drug therapy, drainage of cerebrospinal fluid, or surgical decompression
- Mannitol—improves brain blood flow and lowers intracranial pressure; may worsen bleeding in the brain
- Hypertonic saline (7%)—decreases intracranial pressure; may be used instead of mannitol; may worsen fluid build-up in the brain (cerebral edema) if it gets outside of the blood vessels
- Medication to remove excess fluid from the body (known as a “diuretic”), such as furosemide—decreases cerebrospinal fluid production; lowers intracranial pressure; used in patients with bleeding, congestive heart failure, and kidney failure characterized by the lack of production of urine (known as “anuric kidney failure”); use before mannitol or as sole diuretic
- Steroids (high-dose methylprednisolone)—no benefit in acute management of brain injury in people; no improvement on long-term outcome
- Provide analgesia/sedatives, as indicated
- Prevent thrashing, seizures, or uncontrolled motor activity—such activity may elevate intracranial pressure; diazepam may be required
- Consider loading dosage of phenobarbital, if seizure activity is present
- Desmopressin (DDAVP) for cases with high levels of sodium in the blood that does not respond to medical treatment (known as “refractory hyponatremia”); desmopressin is a synthetic antidiuretic hormone (ADH), the hormone that decreases the amount of water in the urine and thus, maintains hydration of the body
- Medically induced (barbiturate) coma—for increased intracranial pressure that does not respond to treatment to lower metabolism in the brain; must pass an endotracheal tube into the windpipe or trachea (intubate) and support blood pressure, oxygenation, and breathing

Other

- Cooling the patient down to 32° to 33° C (89° to 91° F) may provide brain protection, when done within 6 hours of severe brain injury
- Glucose supplementation—as required for low levels of glucose (sugar) in the blood (hypoglycemia)
- Insulin—as required for high levels of glucose (sugar) in the blood (hyperglycemia); requires close monitoring of blood glucose
- Cisapride may be necessary to promote gastrointestinal motility; cisapride is a drug that improves the movement of contents through

the stomach and intestines (known as a “gastrointestinal prokinetic agent”)



FOLLOW-UP CARE

PATIENT MONITORING

- Repeated nervous system examinations—to detect deterioration of function that warrants aggressive therapeutic intervention
- Blood pressure—to keep fluid therapy adequate for blood flow to the brain, but avoid high blood pressure (hypertension)
- Blood gases (measurements of oxygen and carbon dioxide levels in arterial blood)—to assess need for oxygen supplementation or ventilation
- Blood glucose—maintain at 80 to 120 mg/dl
- Electrocardiogram (“ECG,” a recording of the electrical activity of the heart)—to detect irregular heartbeats (known as “arrhythmias”) that may affect blood flow and oxygen levels in the brain
- Intracranial pressure—to detect significant elevations; monitor success of treatment

PREVENTIONS AND AVOIDANCE

- Keep pets in a confined area with supervised activity (avoid trauma and exposure to poisons)

POSSIBLE COMPLICATIONS

- Increasing intracranial pressure
- Brain pushes downward in the skull and herniates through the opening that leads to the neck (known as “tentorial herniation” or “brain herniation”), leading to death
- Bleeding into the skull (intracranial hemorrhage)
- Progression of signs, indicating deterioration of brain injury
- Seizures
- Malnutrition
- Lung congestion (secondary to lying down)
- Drying of the corneas (the clear outer layer of the front of the eye)
- Skin lesions that develop due to contact with urine, when the hair and skin remain damp (known as “urine scald”)
- Airway blockage from accumulation of mucus
- Irregular heartbeats (arrhythmias)—usually involves a slow heart rate (known as “bradyarrhythmias”)
- Low blood pressure (hypotension)
- Increased levels of sodium in the blood (hypernatremia)
- Decreased levels of potassium in the blood (known as “hypokalemia”)
- Breathing failure
- Death

EXPECTED COURSE AND PROGNOSIS

- Minimal primary brain injury and secondary injury consisting of fluid build-up in the brain (cerebral edema)—best prognosis
- No deterioration of nervous system status for 48 hours—better prognosis
- Rapid resuscitation of systolic blood pressure to greater than 90 mmHg—better outcome
- Maintenance of blood glucose at 80 to 120 mg/dl associated with better outcome in people



KEY POINTS

- The extent of brain recovery may not be evident for several days; and may be more than 6 months for residual nervous system deficits
- Serious generalized (systemic) abnormalities may contribute to the instability of the nervous system

BRAIN TUMORS



BASICS

OVERVIEW

- Brain tumors may be classified as “primary” or “secondary”
- “Primary brain tumors” originate from cells normally found within the brain and meninges (membranes covering the brain)
- “Secondary tumors” are either cancer that has spread to the brain (known as “metastasis”) from a primary tumor outside the nervous system, or tumors that affect the brain by invading or extending into brain tissue from adjacent non-nervous system tissues, such as bone
- Pituitary gland tumors (adenomas or carcinomas) and tumors arising from cranial nerves are considered secondary brain tumors; the “cranial nerves” are nerves that originate in the brain and go to various structures of the head (such as the eye, face, and tongue)
- Brain tumors appear to be more common in dogs than in other domestic species

GENETICS

- An unusually high incidence of benign tumors originating from the membranes covering the brain (membranes are the meninges; tumors are “meningiomas”) has been reported in cats with mucopolysaccharidosis type I; “mucopolysaccharidosis” is the term for a group of inherited disorders in which particular enzymes necessary for normal cell function (that is, metabolism) are deficient

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Meningiomas (benign tumors originating from the membranes covering the brain) occur most frequently in dolichocephalic breeds of dog; “dolichocephalic breeds” are dogs that have long heads and noses, such as the collie and Afghan hound
- Glial cell tumors and pituitary tumors occur commonly in short-nosed, flat-faced (known as “brachycephalic”) breeds of dog; “glial cell tumors” originate from cells that surround and support nerve cells and act as insulation between these cells
- Canine breeds that appear to be more likely to develop brain tumors than other breeds include the boxer, golden retriever, Doberman pinscher, Scottish terrier, and Old English sheepdog
- No increased likelihood of developing brain tumors has been identified in any breed of cat

Mean Age and Range

- Brain tumors occur in dogs and cats of any age
- Most frequent in older dogs, with the greatest incidence in dogs greater than 5 years of age

Predominant Sex

- Older male cats appear to be most likely to develop meningiomas (benign tumors originating from the membranes covering the brain)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary with tumor location
- Most frequently recognized clinical sign associated with a brain tumor of a dog or cat is seizures, particularly if the first seizure occurs after the animal has reached 5 years of age
- Other clinical signs frequently associated with a brain tumor are abnormal behavior and mental status; vision abnormalities (such as blindness); circling; wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”); head tilt; being overly sensitive to pain or touch (known as “hyperesthesia”) in the area of the neck

CAUSES

- Uncertain
- Dietary, environmental, genetic, chemical, viral, traumatic, and immune system factors may be considered

RISK FACTORS

- Uncertain



TREATMENT

HEALTH CARE

- The major goals of therapy for a brain tumor are to control secondary effects, such as increased pressure of the cerebrospinal fluid within the skull cavity (known as “increased intracranial pressure”) or fluid build-up in the brain (known as “cerebral edema”), and to eradicate the tumor or reduce its size
- Three methods of therapy for a brain tumor are available at this time for use in dogs and cats including surgery, radiation therapy, and

chemotherapy

Surgery

- Neurosurgery for complete surgical removal, partial removal, or biopsy of the brain tumor
- Meningiomas (benign tumors originating from the membranes covering the brain) may be able to be removed completely (or almost completely) by means of surgery, especially in cats

Radiation Therapy

- Radiation therapy may be used either alone or in combination with other treatments for either primary or secondary brain tumors
- Careful treatment planning by an experienced radiation therapist is essential to the success of radiation therapy

Chemotherapy

- Chemotherapy drugs (such as carmustine [BCNU] or lomustine [CCNU]) may result in reduction of tumor size, and in improvement of clinical signs in dogs with glial cell tumors; “glial cell tumors” originate from cells that surround and support nerve cells and act as insulation between these cells
- Cytosine arabinoside (ARA-C) has been used in dogs to treat lymphoma of the central nervous system; “lymphoma” is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids may be utilized to decrease fluid build-up (edema) and, in some cases (such as for treatment of lymphoma), to slow tumor growth; “lymphoma” is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body
- Some animals with brain tumors will have dramatic improvement in clinical signs for weeks or months with sustained steroid treatment
- Medications to control seizures (known as “anticonvulsants”), such as phenobarbital or bromide
- Mannitol to reduce increased intracranial pressure (increased pressure of the cerebrospinal fluid within the skull cavity)



FOLLOW-UP CARE

PATIENT MONITORING

- Serial nervous system examinations
- Serial diagnostic imaging (computed tomography [CT or CAT scan], magnetic resonance imaging [MRI])

POSSIBLE COMPLICATIONS

- Aspiration pneumonia due to depressed swallowing reflexes associated with increased intracranial pressure (increased pressure of the cerebrospinal fluid within the skull cavity)
- Seizures

EXPECTED COURSE AND PROGNOSIS

- Information is limited; however, prognosis generally is guarded to poor for animals treated to control the secondary effects of a brain tumor only, without an attempt to eradicate the tumor; the results of one study indicate a mean and median survival of 81 days and 56 days, respectively, following CAT scan diagnosis of a primary brain tumor in 8 dogs
- Several studies confirm that the prognosis for a dog or cat with a primary brain tumor may be improved significantly by surgical removal of the tumor, radiation therapy, and chemotherapy (used either alone or in combination)



KEY POINTS

- Brain tumors may be classified as “primary” or “secondary”
- “Primary brain tumors” originate from cells normally found within the brain and meninges (membranes covering the brain)
- “Secondary tumors” are either cancer that has spread to the brain (known as “metastasis”) from a primary tumor outside the nervous system, or tumors that affect the brain by invading or extending into brain tissue from adjacent non-nervous system tissues, such as bone
- Three methods of therapy for a brain tumor are available at this time for use in dogs and cats including surgery, radiation therapy, and chemotherapy
- Prognosis generally is guarded to poor for animals treated to control the secondary effects of a brain tumor only, without an attempt to eradicate the tumor
- Several studies confirm that the prognosis for a dog or cat with a primary brain tumor may be improved significantly by surgical

removal of the tumor, radiation therapy, and chemotherapy (used either alone or in combination)

DIFFICULTY BREATHING, RAPID BREATHING, PANTING



BASICS

OVERVIEW

- Difficulty breathing (known as “dyspnea”)—a subjective term that in human medicine means “an uncomfortable sensation in breathing” or a sensation of air hunger; in veterinary medicine, it means difficulty breathing or respiratory distress
- Rapid breathing (known as “tachypnea”) is an increased respiratory rate
- Panting is rapid, shallow, open-mouth breathing

SIGNALMENT/DESCRIPTION of ANIMAL

- Dogs and cats; no breed, age, or sex predilection

SIGNS/OBSERVED CHANGES in the ANIMAL

- Patients with primary respiratory disease—coughing, rapid breathing (tachypnea), exercise intolerance
- Nonrespiratory causes—signs associated with the primary disease
- General signs of dyspnea—increased abdominal effort when breathing; nasal flaring (esp. cats); open-mouth breathing; neck extension; moving the elbows out, away from the body to an attempt to increase lung capacity (elbow abduction)
- Nasal disease—noisy breathing when inhaling (known as “stertor”); lack of airflow through nostrils; difficulty breathing improves with open-mouth breathing
- Upper airway disease (involving nose, throat and/or windpipe, so called “upper airway”)—high-pitched, noisy breathing (known as “stridor”); increased body temperature (known as “hyperthermia”)
- Dynamic obstruction, such as paralysis of the larynx—difficulty breathing on inhalation or inspiration
- Fixed obstruction, such as a mass or foreign body—difficulty breathing on inhalation or inspiration and exhalation or expiration
- Collapse of the windpipe (trachea)—honking cough
- Lower airway disease (involving the windpipe within the chest, bronchi and lungs, so called “lower airway”)—cough; wheezes heard on listening to the lungs (auscultation) on exhalation or expiration; increased abdominal effort when breathing
- Lung disease—may have crackles heard on listening to the lungs (auscultation); may have normal lung sounds
- Pneumonia—fever
- Heart-related build-up of fluid in the lungs (known as “pulmonary edema”)—heart murmur; low body temperature (known as “hypothermia”); pale gums and moist tissues of the body (mucous membranes); the pink color of the gums is slow to return when the gums are blanched by finger pressure (known as “poor capillary refill time”)
- Diseases involving the space between the chest wall and the lungs (known as “pleural-space disease”)—diminished breath sounds
- Chest-wall disease—visible trauma
- Nonrespiratory diseases—findings will depend on the other diseases, such as pale gums if the animal has a low red-blood cell count (anemia)
- Blood clots to the lungs (known as “pulmonary thromboembolism”)—may have clinical signs of the underlying disease leading to the formation of the blood clots
- Other signs will pertain to the underlying disease, such as shock, trauma

CAUSES & RISK FACTORS

Difficulty Breathing (Dyspnea)

- **Upper Airway Disease** (involving nose, throat and/or windpipe in the neck region, so called “upper airway”)
 - Disorders of the nasal passages leading to blockage of the airways (nasal obstruction)—narrowed nostrils (known as “stenotic nares”); infection; inflammation; cancer; trauma; blood-clotting disorders (known as “coagulopathy”) or bleeding disorders
 - Disorders of the throat (pharynx)—overly long soft palate; polyp in the throat or pharynx (known as “pharyngeal polyp,” seen in cats); turning inside-out of a portion of the voice box or larynx (known as “everted laryngeal sacculles”), such that the space for air to pass through the larynx is decreased; foreign body; cancer
 - Disorders of the voice box (larynx)—paralysis of the larynx; fluid build-up of the laryngeal tissues (edema); collapse of the voice box or larynx (known as “laryngeal collapse”); foreign body; cancer; trauma
 - Disorders of the windpipe (trachea)—windpipe collapse (known as “tracheal collapse”) within the neck; narrowing of the windpipe (known as “tracheal stenosis”); trauma; foreign body; cancer; parasites
- **Lower Airway Disease** (involving the windpipe within the chest, bronchi and lungs, so called “lower airway”)
 - Windpipe (trachea) collapse within the chest
 - Squeezing or compression of lung tissue secondary to enlarged lymph nodes, enlarged left atrium (one of the chambers of the heart), or heart-based tumors
- **Small Airway Disease** (involving the bronchi, bronchioles, and alveoli of the lungs)
 - Allergy; inflammation (such as bronchitis); infection (for example, *Mycoplasma*); parasites; cancer (bronchogenic carcinoma)
- **Lung Tissue (Pulmonary Parenchymal) Disease** (involving the lung tissue or cells)
 - Fluid build-up (edema)—heart-related (cardiogenic) or non-heart related (non-cardiogenic)
 - Pneumonia—infectious; parasitic; aspiration; cancer (primary or metastatic)
 - Inflammatory disease—allergic; infiltrative eosinophilia (characterized by the presence of a large number of

eosinophils [a type of white blood cell] in the lung tissue); acute respiratory distress syndrome; uremic pneumonitis (inflammation of the lung tissue secondary to the presence of urea and other nitrogenous waste products in the blood)

- Bleeding (hemorrhage)—trauma; blood-clotting disorders (coagulopathy)
- Blood clots in the lungs (known as “pulmonary thromboembolism”): immune-mediated breakdown of red blood cells (known as “hemolytic anemia”); heartworm disease; excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); abnormal blood clotting (known as “disseminated intravascular coagulopathy” or “DIC”)

● **Pleural Space Disease** (involving the space between the chest wall and the lungs)

- Air in the pleural space (known as “pneumothorax”)—trauma; secondary to lung tissue (pulmonary parenchymal) disease; ruptured bubble-like areas (known as “bullae”) in the lungs; migrating foreign body; spontaneous
- Fluid in the pleural space (known as “pleural effusion”)—pus; secondary to congestive heart failure; cancer; twisting of a lung lobe (known as “lung-lobe torsion”)
- Blood in the pleural space (known as “hemothorax”)—trauma; clotting disorder (coagulopathy); twisting of a lung lobe (lung-lobe torsion); spontaneous bleeding of the thymus
- Milky fluid in the pleural space (known as “chylothorax”)—unknown reasons (known as “idiopathic”); congestive heart failure; trauma
- Presence of masses or abdominal organs in the pleural space—cancer; secondary to a tear or abnormal opening in the diaphragm (diaphragmatic hernia) through which abdominal contents enter the chest

● **Chest Wall Disease**

- Trauma; cancer; paralysis due to botulism; nervous system disease

● **Abdominal Distention** (putting pressure on the chest and lungs)

- Enlarged organs—enlargement of an organ due to an increase in the number of normal cells (known as “hyperplasia”); cancer; pregnancy; obesity; fluid build-up in the abdomen (known as “ascites”); accumulation of air in the stomach with or without twisting of the stomach (known as “bloat” or “gastric dilatation” or “gastric dilatation/torsion” or “gastric dilatation/volvulus”)

Rapid Breathing (Tachypnea)

- Low levels of oxygen in the blood (known as “hypoxemia”); high levels of carbon dioxide in the blood (known as “hypercapnia”); low blood pressure (hypotension); fever; low levels of red-blood cells (anemia); acidosis (a condition in which levels of acid are increased in the blood); inflammation
- Airway disorders—inhaled irritants; allergic disease; narrowing of the bronchi (known as “bronchoconstriction”); airway squeezing or compression; airway infection
- Interstitial disorders—the interstitium is defined as the small spaces between tissues or organs, interstitial disorders refers to medical conditions located in these spaces—fluid build-up (edema); bleeding (hemorrhage); inflammation; cancer

Panting

- Pain; anxiety; drug therapy (such as with opioids); normal body-heat regulation (that is, panting to cool off body temperature); can be a normal behavioral pattern in some dogs



TREATMENT

HEALTH CARE

- Inpatient care until the cause is identified and treated or determined not to be life-threatening; therapy based on underlying cause
- Oxygen should be administered until patient’s ability to oxygenate is determined
- Keep patient lying on chest (that is, not on its side) until stabilized; and turn hips every 3 to 4 hours
- Upper airway disease—moderately affected animals may benefit from sedation to reduce effort upon inhaling; actively cool patients as necessary since elevated body temperature (hyperthermia) will increase breathing effort; severe upper airway disease may require passing of a tube through the mouth and into the windpipe (intubation) to stabilize the patient; if the problem cannot be resolved immediately, placement of a temporary surgical opening into the windpipe (tracheostomy) may be necessary; remove foreign bodies; perform surgical removal/biopsy of masses, surgical correction for paralysis of the voice box or larynx (laryngeal paralysis) and for the partial upper airway obstruction in short-nosed, flat-faced (brachycephalic) breeds of dogs and cats (known as “brachycephalic airway syndrome”); anti-inflammatory medications for fluid build-up in the voice box or larynx (laryngeal edema)
- Lower airway disease—drugs to enlarge or dilate the bronchii (known as “bronchodilators,” such as terbutaline, theophylline); oxygen therapy until stable; systemic steroids may be necessary to stabilize cats with acute narrowing or decrease in size of the opening of the bronchi (known as “bronchoconstriction”)
- Lung tissue (pulmonary parenchymal) disease—oxygen therapy; antibiotics, if pneumonia; treat blood-clotting disorders; heart-related fluid build-up (known as “cardiogenic edema”) requires diuretics with or without drugs to cause the blood vessels to dilate (known as “vasodilators”); fluid build-up related to non-heart causes (known as “noncardiogenic edema”) requires oxygen therapy; diuretics may be beneficial; use of a hand-controlled bag attached to the airway to push air into the airway or a ventilator (known as “positive-pressure ventilation”) may be necessary if oxygen therapy alone is not adequate to stabilize the patient
- Pleural-space disease—chest tap (known as a “thoracocentesis”) to remove air and/or fluid from the space between the lungs and chest wall—remove as much as possible; chest tube, if repeated chest taps are needed to keep patient stable
- Chest-wall paralysis—positive-pressure ventilation may be necessary, if patient has high levels of carbon dioxide in the blood (known as “hypercapnia”)

- Abdominal distention—drain fluid (ascites) as needed to keep the patient comfortable; relieve stomach distention (as in “bloat”)
- Nonrespiratory diseases—treat primary problem
- Oxygen therapy may be provided via an oxygen cage; a delivery device into the nose (nasal cannula); by making an oxygen “tent” with an Elizabethan collar covered in plastic wrap; an oxygen mask; or allowing oxygen to flow-by the animal’s face—oxygen should be humidified, if administered for more than a few hours

ACTIVITY

- Strict cage confinement until difficulty breathing is resolved

DIET

- Weight-reducing diet, if obesity is contributing to the difficulty in breathing

SURGERY

- Carefully tailor anesthesia to the patient; securing an airway is essential and the ability to use positive-pressure ventilation often is necessary
- Surgery for diaphragmatic hernias or spontaneous pneumothorax (free air in the space between the lungs and chest wall)
- Chest-wall disease—surgery as indicated, particularly if open-chest wound is present



MEDICATIONS

- Varies with underlying cause



FOLLOW-UP CARE

PATIENT MONITORING

- Patients receiving oxygen therapy can be monitored by assessing the degree of breathing difficulty; measure arterial-blood gases
- Pulse oximetry is effective for monitoring patients breathing room air; allowing the patient to try breathing room air (room-air trial) can be useful in evaluating the animal’s response to treatment, degree of breathing difficulty and ability to oxygenate blood (by measuring blood gases)
- Repeat X-rays often are indicated in assessing pulmonary parenchymal disease and pleural-space disease
- Monitor body temperature regularly, as animals with difficult breathing often develop high body temperatures (that is, become hyperthermic), and the increased body temperature (hyperthermia), in turn, will worsen the breathing difficulty (dyspnea)

PREVENTIONS AND AVOIDANCE

- Depends on underlying cause
- Weight reduction, if obesity is contributing to the difficulty in breathing

POSSIBLE COMPLICATIONS

- Breathing difficulties and resulting low oxygen levels in the blood (known as “hypoxia”) can be life threatening; animals with signs of difficult breathing should be evaluated by a veterinarian as soon as possible; animals with severe breathing difficulties should be considered an emergency

EXPECTED COURSE AND PROGNOSIS

- Depends on underlying cause



KEY POINTS

- Breathing difficulties and resulting low oxygen levels in the blood (known as “hypoxia”) can be life threatening; animals with signs of difficult breathing should be evaluated by a veterinarian as soon as possible; animals with severe breathing difficulties should be considered an emergency

CHRONIC BRONCHITIS

(CHRONIC OBSTRUCTIVE PULMONARY DISEASE OR “COPD”)



BASICS

OVERVIEW

- The respiratory tract consists of the “upper respiratory tract” (the nose, nasal passages, throat, and windpipe [trachea]) and the “lower respiratory tract” (the bronchi, bronchioles, and alveoli [the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged])
- The bronchi and bronchioles are considered to be “small airways”
- “Chronic bronchitis” is long-term (chronic) coughing for 2 consecutive months that is not related to another cause (such as cancer; congestive heart failure [CHF], a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs; or infection of the lower respiratory tract)
- Non-reversible and often slowly progressive
- Also known as “chronic obstructive pulmonary disease” or “COPD”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dogs—small- and toy-breed dogs common; also observed in large-breed dogs
- West Highland white terriers—develop a progressive disorder characterized by long-term (chronic) coughing, breathing distress, and short, rough snapping sounds (known as “crackles”) heard when listening to the chest with a stethoscope; also may have “pulmonary fibrosis,” characterized by excessive fibrous or scar-type tissue as part of a reactive process in the lungs
- Cocker spaniels—dilation of bronchi or bronchioles, as a consequence of inflammation or blockage of the airway (known as “bronchiectasis”), common after a history of long-term (chronic) inflammation of the bronchi (bronchitis)
- Cats—Siamese and domestic shorthair

Mean Age and Range

- Most often affects middle-aged and old animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Coughing—hallmark of irritation of the windpipe (trachea) and bronchi (known as “tracheobronchial irritation”); usually “dry” cough; gagging common after coughing (gagging response often misinterpreted as vomiting)
- Exercise intolerance
- Bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”) and even fainting (known as “syncope”) may be noted
- Patients usually are bright and alert with normal body temperature (that is, animal does not have a fever)
- Feeling or applying pressure on the windpipe (trachea) typically results in coughing, because of tracheal sensitivity
- Small airway disease—assumed when an abdominal “push” (during quiet breathing) is noted when the animal breathes out (expiration) or squeaking or whistling sounds (known as “wheezing”) are detected at the end of breathing out
- Heart—heart murmurs common, but not always associated with congestive heart failure; chronic bronchitis usually results in a normal or slower than normal resting heart rate and pronounced irregular heart beat
- Obesity—common; important complicating factor
- Severe dental disease may increase the likelihood of lower airway infection and potential generalized bacterial infection (sepsis)

CAUSES

- Long-term (chronic) airway inflammation initiated by multiple causes

RISK FACTORS

- Recurrent bacterial infection
- Long-term exposure to inhaled irritants
- Obesity
- Dental disease and disease of the voice box (larynx) disease—result in bacterial showering of the lower airways



TREATMENT

HEALTH CARE

- Usually outpatient—oxygen can be set-up to be given at home in some cases
- Inpatient—if patient requires oxygen therapy, injectable medication, or administration of medication in a fine spray or mist that is breathed in (known as “aerosol therapy”); patients that owners cannot keep calm at home during recovery

ACTIVITY

- Exercise—moderate (not forced); useful in clearing secretions from the airways; assists with weight loss
- Limit if exertion causes excessive coughing
- Use a harness instead of a collar

DIET

- Weight loss is critical—improves oxygen levels in the blood, attitude, and exercise tolerance and decreases cough frequency in obese patients

SURGERY

- Treat severe dental disease to minimize secondary bacterial complications



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Steroids

- Decrease airway inflammation and coughing, regardless of the underlying cause
- Indicated for noninfectious causes of long-term inflammation of the bronchi and bronchioles (chronic bronchitis)
- With allergic or hypersensitivity reactions—require long-term administration; attempt to wean off steroids or determine lowest effective dosage

Medications to Enlarge or Dilate the Bronchi (known as “bronchodilators”)

- Beneficial effects (depends on the particular medication)—enlargement or dilation of the bronchi (known as “bronchodilation”); improved secretion clearance mechanism of the lungs (known as “mucociliary clearance”); improvement in function of the diaphragm, the muscle between the chest and abdomen; lowered blood pressure in the pulmonary artery
- β -agonists—terbutaline and albuterol
- Sustained-release theophylline—oral administration
- Aminophylline—immediate-release tablets or injectable formulations are not recommended

Antibiotics

- Select on the basis of bacterial culture and sensitivity test results
- While waiting for bacterial culture and sensitivity results—antibiotic that is effective against gram-negative bacteria (antibiotics such as potentiated sulfa/trimethoprim, amoxicillin/clavulanic acid, or enrofloxacin)
- Associated long-term (chronic) aspiration pneumonia or dental disease—may prefer an antibiotic that is effective against bacteria that can live and grow in the absence of oxygen (known as “anaerobic bacteria”) and gram-positive bacteria

Medications to Control Coughing (known as “antitussives”)

- Indicated for cough that is nonproductive (that is, a “dry” cough in which no sputum [secretion or material] is coughed up); paroxysmal (that is, coughing episodes occur suddenly at fairly regular intervals); continuous (that is, the coughing goes on without letting up); or debilitating (that is, the cough is severe enough to affect the animal in general, even leading to lack of sleep and to weakness)
- Dogs—butorphanol; hydrocodone; codeine
- Metered dose inhalers (steroids—Flovent®; medications to enlarge or dilate the bronchi [bronchodilators]—albuterol) and aerosolized antibiotics (gentocin) may be administered via face mask
- Serotonin blockade (but not leukotriene blockers)—may block airway “hyper-responsiveness” in cats with airway disease
- Cyclosporine-induced immune suppression—cyclosporine is a medication that decreases the immune response; may block structural changes in airways associated with asthma in cats



FOLLOW-UP CARE

PATIENT MONITORING

- Follow abnormalities revealed by physical examination and selected diagnostic tests—determine response to treatment
- Monitor weight and arterial blood gases (measurements of oxygen and carbon dioxide levels in arterial blood)—usually improve after significant weight loss

PREVENTIONS AND AVOIDANCE

- Avoid and address/correct risk factors

POSSIBLE COMPLICATIONS

- Fainting (syncope)—frequent complication of long-term (chronic) coughing, particularly in toy-breed dogs
- Increased blood pressure in the lungs (known as “pulmonary hypertension”) and heart disease secondary to lung disease (known as “cor pulmonale”)—most serious complications

EXPECTED COURSE AND PROGNOSIS

- Progressive airway changes—fainting (syncopal) episodes; long-term (chronic) low levels of oxygen in the blood and body tissues (known as “hypoxia”); enlargement of the right ventricle (a chamber of the heart; condition known as “right ventricular hypertrophy”); and increased blood pressure in the lungs (pulmonary hypertension)
- Sudden (acute) worsening of signs—common with seasonal changes, air quality changes, worsened inflammation, and potentially the development of secondary infection



KEY POINTS

- Chronic bronchitis is an incurable disease and complete suppression of all coughing is an unattainable goal
- Aggressive treatment—including weight control, avoiding risk factors, and medical treatment—minimizes the severity of the coughing and slows disease progression in most patients

DISEASE CAUSED BY *BRUCELLA*, A TYPE OF BACTERIA (BRUCELLOSIS)



BASICS

OVERVIEW

- Contagious disease of dogs caused by *Brucella canis*, a small, intracellular, gram-negative bacteria
- Characterized by abortion and infertility in females and inflammation of the epididymis (where sperm are stored prior to ejaculation; condition known as “epididymitis”) and wasting or decrease in size of the testicles (known as “testicular atrophy”) in males
- Dogs may become infected during breeding or through contact with aborted materials or vaginal discharge following abortion
- A female dog is a “bitch”

GENETICS

- No known genetic susceptibility to developing brucellosis
- Occurs most commonly in beagles

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and, infrequently, people

Breed Predilections

- No evidence of breed susceptibility, but exceptionally high number of cases in beagles
- Infected Labrador retrievers and several other breeds found in commercial kennels (“puppy mills”)

Mean Age and Range

- No age preference
- Most common in sexually mature dogs

Predominant Sex

- Both sexes are affected
- More common in females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Suspect whenever a bitch experiences abortions or reproductive failures or a male has genital disease
- Affected animals, especially females, may appear healthy or have vague signs of illness
- Sluggishness (lethargy)
- Loss of libido
- Back pain
- Abortion—commonly at 6 to 8 weeks after conception, although pregnancy may terminate at any stage
- Males—swollen scrotal sacs, often with inflammation of the skin covering the scrotum (known as “scrotal dermatitis”); enlarged and firm epididymides (plural of epididymis; where sperm are stored prior to ejaculation)
- Long-term (chronic) infection—wasting or decrease in size of one or both testicles (known as “testicular atrophy”); cloudy eyes (inflammation of the front part of the eye, including the iris [known as “anterior uveitis”] with fluid build-up in the clear part of the eye [known as “corneal edema”]); spinal pain; weakness in the hindquarters; wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)
- Fever (rare)
- Enlarged lymph nodes (common)
- Vaginal discharge, may last for several weeks after an abortion

CAUSES

- *Brucella canis*—gram-negative bacteria

RISK FACTORS

- Breeding kennels and pack hounds
- Risk increases when popular breeding animals become infected
- Contact with strays in a particular region (known as “endemic area”) where *Brucella* is present



TREATMENT

HEALTH CARE

- Outpatient

ACTIVITY

- Restrict working dogs

SURGERY

- Neutering/spaying plus medical treatment—when euthanasia is unacceptable to an owner



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Several therapeutic regimens have been evaluated, but results have been equivocal
- Most successful treatment—combination of a tetracycline (tetracycline hydrochloride, chlortetracycline, or minocycline) or doxycycline and dihydrostreptomycin
- Enrofloxacin recently reported to be effective
- Gentamicin—limited success; insufficient data on effectiveness when combined with tetracycline



FOLLOW-UP CARE

PATIENT MONITORING

- Serologic tests (blood tests that detect the presence of antibodies to a certain disease-causing agent or antigen; an “antibody” is a protein that is produced by the immune system in response to a specific antigen)—monthly for at least 3 months after completion of treatment; continuous, persistent decline in antibodies to negative status (known as a “seronegative status”) indicates successful treatment
- Infection becoming active again (indicated by a rise in antibody levels on serologic tests and recurrence of bacteria in the blood [known as “bacteremia”] after treatment)—re-treat; spay or neuter and re-treat; or euthanize
- Bacterial blood cultures—negative for at least 3 months after completion of treatment

PREVENTIONS AND AVOIDANCE

- Vaccine—none; would complicate serologic testing (blood tests that detect the presence of antibodies to a certain disease-causing agent or antigen; an “antibody” is a protein that is produced by the immune system in response to a specific antigen)
- Testing—all brood bitches, before they come into “heat” or “estrus,” if a breeding is planned; males used for breeding, at frequent intervals
- Quarantine and test all new dogs twice at monthly intervals before allowing them to enter a breeding kennel

POSSIBLE COMPLICATIONS

- Owners may be reluctant to spay or neuter or destroy valuable dogs, regardless of treatment failure
- Infertility
- Human exposure/infection

EXPECTED COURSE AND PROGNOSIS

- Prognosis is guarded
- If infected for less than 3 to 4 months—likely to respond to treatment
- Long-term (chronic) infections—males may fail to respond to treatment
- Successfully treated (decline in antibodies to negative status [seronegative status]) dogs—fully susceptible to reinfection



KEY POINTS

- Goal of treatment is the eradication of *Brucella canis* from the animal (as indicated by a decline in antibodies to negative status [seronegative status] and no bacteria in the blood [bacteremia] for at least 3 months), but sometimes the result of treatment is persistent low antibody titers, with no generalized (systemic) infection
- Antibiotic treatment, especially minocycline and doxycycline, is expensive, time-consuming, and controversial (because outcomes are uncertain)
- Treatment is not recommended for breeding or commercial kennels; it is recommended only for non-breeding dogs or those that have

been spayed or neutered

- Before treatment is attempted for an intact household pet or breeding dog, the client must clearly agree that the animal must be neutered or destroyed if treatment fails
- Owners must understand the ethical considerations and obligations not to sell or distribute infected dogs
- Zoonotic potential of brucellosis should be considered; a “zoonosis” is a disease that can be passed from animals to people

DILATED CARDIOMYOPATHY IN DOGS

(A TYPE OF HEART-MUSCLE DISEASE)



BASICS

OVERVIEW

- The heart of the dog is composed of four chambers; the top two chambers are the left and right atria and the bottom two chambers are the left and right ventricles; heart valves are located between the left atrium and the left ventricle (mitral valve); between the right atrium and the right ventricle (tricuspid valve); from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve); and from the right ventricle to the main pulmonary (lung) artery (pulmonary valve)
- “Cardiomyopathy” is the medical term for disease of the heart muscle; “dilated cardiomyopathy” or “DCM” is a disease in which the heart muscle is flabby and weak
- “Dilated cardiomyopathy” in dogs is characterized by left- and right-sided enlargement of the lumen of the chambers of the heart; normal coronary arteries; normal (or minimally diseased) atrioventricular valves (that is, the mitral and tricuspid valves); significantly decreased ability to contract the heart muscle; and heart-muscle dysfunction

GENETICS

- Genetic cause or heritable susceptibility strongly suspected in many breeds and documented in some breeds, with variable forms of inheritance

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Doberman pinscher, boxer
- “Giant” breeds: Scottish deerhound, Irish wolfhound, Great Dane, Saint Bernard, Afghan hound, Bernese mountain dog
- Cocker spaniel, Portuguese water dog

Mean Age and Range

- 4 to 10 years of age

Predominant Sex

- Males are more likely to be affected than females in most, but not all, breeds

SIGNS/OBSERVED CHANGES in the ANIMAL

- Some dogs do not have clinical signs, having what is termed “preclinical dilated cardiomyopathy”
- Rapid breathing (known as “tachypnea”), difficulty breathing (known as “dyspnea”), coughing
- When listening to the chest with a stethoscope, may hear muffled breath sounds due to the presence of fluid between the chest wall and lungs (known as “pleural effusion”) or may hear short, rough snapping sounds (known as “crackles”) due to the presence of fluid in the lungs (known as “pulmonary edema”)
- Weight loss
- Weakness, sluggishness (lethargy), lack of appetite (known as “anorexia”)
- Abdominal swelling or distention
- Fainting (known as “syncope”)
- Depression
- Possible cardiogenic shock (condition in which the heart is unable to pump adequate blood to the tissues and the tissues become oxygen starved)
- Abnormal femoral pulses from the low volume of blood being pumped by the heart (known as “cardiac output”)
- Irregular heart beats (arrhythmias)
- Pulse deficits with irregular or rapid heart beats (such as seen with atrial fibrillation, ventricular or supraventricular premature contractions, and paroxysmal ventricular tachycardia); the “pulse” is the rhythmic “throbbing” of the arteries as the heart beats—normally the artery “throbs” each time the heart beats so that the pulse and the heart rate are the same; pulse deficits occur when the pulse and heart rate do not match, with the number of pulses being lower than the number of heart beats—pulse deficits usually indicate serious disease as the heart is unable to pump adequate blood with each heart beat
- The external jugular veins (located on either side of the neck) may have a pulse from backflow of blood through the tricuspid valve (known as “tricuspid regurgitation”), irregular heart beats (known as “arrhythmias”), or right-sided congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Heart murmur or abnormal heart sounds
- Pink color of the gums is slow to return when the gums are blanched by finger pressure (known as “poor capillary refill time”)
- Bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”)
- Enlarged liver, with or without build-up of fluid in the abdomen (fluid known as “ascites”)

CAUSES

- Primary mechanism yet to be identified and is of unknown cause (so called “idiopathic disease”) in the vast majority of cases
- Majority of cases probably represent familial (runs in certain families or lines of animals) abnormalities of structural or contractile heart proteins
- Nutritional deficiencies (taurine and/or carnitine) have been documented in several breeds, including golden retrievers, boxers, Doberman pinschers, and cocker spaniels; “taurine” is an amino acid (the smallest component of protein); “carnitine” is a compound involved in enzymes that transport fatty acids, which are important in the heart for energy
- Viral, protozoal, and immune-mediated mechanisms have been proposed
- Inadequate thyroid hormone (known as “hypothyroidism”) may cause reversible heart-muscle failure



TREATMENT

HEALTH CARE

- With the exception of severely affected dogs, most therapy can be administered on an outpatient basis
- Identify patient problems: left- or right-sided congestive heart failure, irregular heart beats (arrhythmias), decreased body temperature (known as “hypothermia”), kidney failure, and/or shock

ACTIVITY

- Allow the dog to choose its own level of activity

DIET

- Goal: reduce dietary sodium intake
- Severe sodium restriction is not necessary when using medications to enlarge or dilate the blood vessels (known as “vasodilators”) and to remove excess fluid from the body (known as “diuretics”)
- Best to use commercially prepared diets



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Initial Stabilization

- Treat low levels of oxygen in the blood (known as “hypoxemia”) with oxygen administration; prevent heat loss, if the patient has low body temperature (hypothermia) by placing in a warm environment; administer fluids only after fluid build-up in the lungs (pulmonary edema) is controlled and/or fluid build-up in the space between the chest wall and lungs (pleural effusion) has been drained
- If fluid build-up in the lungs (pulmonary edema) is present: medications to remove excess fluids from the body (diuretics), such as furosemide, should be administered
- 2% topical nitroglycerin for the first 24 to 48 hours for patients with severe fluid build-up in the lungs (pulmonary edema)
- If severe heart failure and cardiogenic shock (condition in which the heart is unable to pump adequate blood to the tissues and the tissues become oxygen starved) are present, dobutamine (drug to increase contraction of the heart muscle) may be indicated; pimobendan may be beneficial as well
- Digoxin (heart medication used in treatment of congestive heart failure and certain irregular heart beats [arrhythmias])
- Other medications (such as lidocaine or procainamide) may be administered for certain irregular heart beats (arrhythmias)

Maintenance Therapy

- Medications to enlarge or dilate blood vessels (vasodilators)—especially the angiotensin-converting enzyme (ACE) inhibitors (such as enalapril, benazepril, lisinopril) are considered a cornerstone of therapy for dilated cardiomyopathy
- Enalapril, benazepril, or lisinopril should be initiated early in treatment
- Other medications to enlarge or dilate blood vessels (vasodilators), including hydralazine and amlodipine; they may be used instead of or in addition to an ACE inhibitor (beware of low blood pressure [known as “hypotension”])
- A daily maintenance dose of digoxin is given to most giant-breed dogs; digoxin is used primarily for control of ventricular response rate in atrial fibrillation (rapid, irregular heart rhythm involving the top two chambers of the heart [atria])
- Pimobendan is used to improve heart-muscle contraction
- Furosemide (a diuretic to remove excess fluid from the body) is used to control fluid build-up in the lungs (pulmonary edema), in the space between the chest wall and lungs (pleural effusion), or in the abdomen (ascites)
- Spironolactone (a diuretic to remove excess fluid from the body) reduces mortality in humans with heart failure
- Beta-blockers can be used cautiously once heart failure is controlled with other drugs; if tolerated, may improve heart-muscle function with long-term (chronic) use
- The role of taurine and carnitine in the therapy of dilated cardiomyopathy remains controversial; however, American cocker spaniels with dilated cardiomyopathy generally respond favorably to taurine and l-carnitine supplementation

Irregular Heart Beats (Arrhythmias)

- In the case of atrial fibrillation (rapid, irregular heart rhythm involving the top two chambers of the heart [atria]), slowing of the ventricular response rate is achieved with long-term (chronic) administration of digitalis combined with atenolol or diltiazem

- Therapeutic goal is obtaining a resting ventricular rate between 100 to 140 beats per minute
- Recent evidence suggests that amiodarone may either control ventricular response rate or in some cases (32%) result in conversion to normal heart rhythm
- Long-term (chronic) oral therapy for fast heart rate originating in the ventricles (known as “ventricular tachycardia”) includes procainamide, mexiletine, amiodarone, or sotalol
- Procainamide and mexiletine can be combined with a beta-blocker, if necessary
- The role of co-enzyme Q10 in the treatment of dilated cardiomyopathy remains to be determined



FOLLOW-UP CARE

PATIENT MONITORING

- Serial clinical examinations, chest X-rays, blood-pressure measurements, routine blood work (serum biochemical evaluations, including electrolytes) and electrocardiograms (“ECGs,” recordings of the electrical activity of the heart) are most helpful
- Repeat echocardiography (use of ultrasound to evaluate the heart and major blood vessels) rarely is informative or indicated
- Serial evaluation of serum digoxin levels (therapeutic range, 0.5 to 1 ng/ml) taken 6 to 8 hours following administration of the pill and serum biochemistries may help prevent side effects of the drug

POSSIBLE COMPLICATIONS

- Sudden death due to irregular heart beats (arrhythmias)
- Side effects of drugs associated with medical management

EXPECTED COURSE AND PROGNOSIS

- Always fatal
- Death usually occurs 6 to 24 months following diagnosis
- Doberman pinschers typically have a worse prognosis, with survival generally less than 6 months from the time of diagnosis (addition of pimobendan in their treatment may increase survival time substantially)



KEY POINTS

- Understand potential signs associated with progression of disease and adverse side effects of medications
- Monitoring resting breathing rate often gives insight into worsening condition

HYPERTROPHIC CARDIOMYOPATHY IN CATS

(TYPE OF HEART MUSCLE DISEASE)



BASICS

OVERVIEW

- “Hypertrophic” refers to hypertrophy; “hypertrophy” is an increase in size of a tissue or organ that is not due to formation of a tumor; “cardiomyopathy” is a disease of heart muscle
- The heart of the cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; the left ventricle pumps blood into the aorta (the main artery of the body) and thus, into the body
- “Hypertrophic cardiomyopathy” is a disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle; the disease occurs independently of other heart or generalized (systemic) disorders
- Also known as “HCM”

GENETICS

- Some families of cats have been identified with a high number of cases of hypertrophic cardiomyopathy, and the disease appears to be inherited as an autosomal dominant trait in Maine coon cats, where a mutation (“MyBPC”) has been identified in at least one large family
- Genetics have not been determined definitively in other families or breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Breed Predispositions

- A familial (runs in certain families or lines of animals) association has been documented in Maine coon cats, American shorthairs, and Persians

Mean Age and Range

- Most common in cats 5 to 7 years of age, with reported age range of 3 months to 17 years
- Most often a disease of young to middle-aged cats
- Unexplained murmurs in senior cats more likely are associated with increased levels of thyroid hormone (known as “hyperthyroidism”) or high blood pressure (known as “hypertension”) than with hypertrophic cardiomyopathy

Predominant Sex

- Male

SIGNS/OBSERVED CHANGES in the ANIMAL

- Difficulty breathing (known as “dyspnea”)
- Lack of appetite (known as “anorexia”)
- Exercise intolerance
- Vomiting
- Collapse
- Sudden death
- Coughing is uncommon in cats with disease of the heart muscle (cardiomyopathy) and usually suggests lung disease
- Abnormal heart sounds when listening to the heart with a stethoscope (examples include gallop rhythm, heart murmurs)
- Muffled heart sounds, lack of chest compliance, and difficulty breathing (dyspnea) characterized by rapid, shallow breathing may be associated with fluid build-up in the space between the lungs and chest wall (known as “pleural effusion”)
- Short, rough snapping breath sounds (known as “crackles”) may be heard when listening to the chest with a stethoscope, if fluid build-up in the lungs (known as “pulmonary edema”) is present
- Weak femoral pulse
- Sudden (acute) hind-limb paralysis with cold limbs, absence of femoral pulse and bluish discoloration (known as “cyanosis”) of the pads and nailbeds in animals with blood clots in the aorta (known as “aortic thromboembolism”)
- Irregular heart beat (known as an “arrhythmia”) in some animals

CAUSES

- Usually unknown—multiple possible causes exist
- Genetics: MyBPC mutation in some cats with hypertrophic cardiomyopathy

RISK FACTORS

- Offspring of animals with familial (runs in certain families or lines of animals) mutation of MyBPC



TREATMENT

HEALTH CARE

- Cats with congestive heart failure should be hospitalized for initial medical management; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs
- Minimize stress
- Oxygen, if cat is having difficulty breathing (dyspnea)
- Warm environment, if cat has low body temperature (known as "hypothermia")

ACTIVITY

- Restricted

DIET

- Sodium restriction in cats with congestive heart failure; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Diltiazem

- Beneficial effects may include slower heart rate, resolution of irregular heart beats (arrhythmias), improved relaxation of the heart muscle during the "rest" phase of the heart beat (known as "diastolic relaxation"), enlargement of heart blood vessels (known as "coronary vasodilation"), enlargement of blood vessels in the body (known as "peripheral vasodilation"), platelet inhibition
- May reduce enlargement (hypertrophy) of the heart muscle in some cats
- Superior to propranolol and verapamil, according to one small study
- Role in patients without clinical signs is unresolved

Beta Blockers

- Atenolol
- Beneficial effects may include slowing of heart rate, correcting irregular heart beats (arrhythmias), platelet inhibition
- More effective than diltiazem in controlling blockage of blood flow as the left ventricle pumps blood into the aorta, the main artery of the body (condition known as "dynamic outflow tract obstruction")
- Role in patients without clinical signs is unresolved, but authors generally use if dynamic outflow obstruction and enlargement (hypertrophy) of the heart muscle are present
- Should not be used in cases of congestive heart failure; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs

Aspirin

- Decreases crowding or massing together (aggregating) of platelets, hopefully minimizing the risk of blood clots (thromboembolism); however, blood clots can still develop despite aspirin administration

Furosemide (Medication to Remove Excess Fluid from the Body {Diuretic})

- Animals with critical difficulty breathing (dyspnea) often require high dosage to stabilize them; indicated to treat fluid build-up in the lungs (pulmonary edema), in the space between the lungs and chest wall (pleural effusion), and in the abdomen (known as "ascites")
- Cats are sensitive to furosemide and prone to dehydration, excess levels of urea and other nitrogenous waste products in the blood (known as "uremia" or "azotemia"), and low levels of potassium in the blood (known as "hypokalemia")
- Once fluid build-up in the lungs (pulmonary edema) resolves, the dosage should be tapered (as directed by your cat's veterinarian) to the lowest dose that controls fluid build-up (edema)

Nitroglycerin Ointment

- Often used in stabilization of cats with severe fluid build-up in the lungs (pulmonary edema) or in the space between the lungs and chest wall (pleural effusion)
- When used intermittently, it may be useful for long-term management of cases that do not respond well to medical treatment

Angiotensin-Converting Enzyme (ACE) Inhibitors

- Enalapril or benazepril
- Indications in cats with hypertrophic cardiomyopathy not well-defined—may be used in cases with congestive heart failure; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs

Spirinolactone (Medication to Remove Excess Fluid from the Body {Diuretic})

- Used in conjunction with furosemide in cats with congestive heart failure; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs

Warfarin (Medication to Prevent Blood Clotting)

- Used sometimes in cats at high risk for developing blood clots (thromboembolism)

Dalteparin (Fragmin®)

- Alternative to warfarin that may eliminate need for patient monitoring for those at high risk of blood clots in the aorta (aortic thromboembolism)

Clopidogrel (Plavix®)

- Platelet function inhibitor, superior to aspirin in people; studies currently under way to evaluate effectiveness in preventing blood clots in the aorta (aortic thromboembolism) in cats

Beta Blocker Plus Diltiazem

- Cats that continue to have a rapid heart rate (known as “tachycardia”) on a single medication can be treated cautiously with a combination of a beta blocker and diltiazem; these cats should be monitored closely for slow heart rate (bradycardia) and low blood pressure (hypotension)



FOLLOW-UP CARE

PATIENT MONITORING

- Observe closely for difficulty breathing (dyspnea), sluggishness (lethargy), weakness, lack of appetite (anorexia), and painful hind-limb weakness or paralysis
- If treating with warfarin, monitor blood work (prothrombin time) to evaluate effectiveness of drug in decreasing likelihood of blood clot and to determine appropriate dose to avoid bleeding
- If treating with an ACE inhibitor or spironolactone, monitor kidney function and electrolytes
- Repeat echocardiogram (use of ultrasound to evaluate the heart and major blood vessels) in 6 months to assess effectiveness of treatment for hypertrophic cardiomyopathy and to evaluate the need for more aggressive treatment to prevent blood clots in the aorta (aortic thromboembolism)

PREVENTIONS AND AVOIDANCE

- Avoid stressful situations that might lead to congestive heart failure; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs

POSSIBLE COMPLICATIONS

- Heart failure
- Blood clot in the aorta (aortic thromboembolism) and hind-limb paralysis
- Irregular heart beats (arrhythmias)/sudden death

EXPECTED COURSE AND PROGNOSIS

- Prognosis varies considerably, probably because multiple causes exist for hypertrophic cardiomyopathy; in one study, cats living at least 24 hours after presentation to a veterinarian had the following survival times:
 - Asymptomatic cats (that is, no clinical signs of hypertrophic cardiomyopathy): median survival time of 563 days (range: 2 to 3778 days)
 - Cats with fainting (known as “syncope”): median survival time of 654 days (range: 28 to 1505 days)
 - Cats with congestive heart failure: median survival time of 563 days (range: 2 to 4418 days)
 - Cats with blood clots in the aorta (aortic thromboembolism): median survival time of 184 days (range 2 to 2278 days)
- Older age of the cat and larger left atrium predicted shorter survival time



KEY POINTS

- Many cats diagnosed while not showing signs of disease eventually develop congestive heart failure and may develop blood clots in the aorta (aortic thromboembolism) and die suddenly; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs
- If cat is receiving warfarin to control the formation of blood clots, minimize potential for trauma and subsequent bleeding

RESTRICTIVE CARDIOMYOPATHY IN CATS

(A TYPE OF HEART-MUSCLE DISEASE)



BASICS

OVERVIEW

- The heart of the cat is composed of four chambers; the top two chambers are the left and right atria and the bottom two chambers are the left and right ventricles; heart valves are located between the left atrium and the left ventricle (mitral valve); between the right atrium and the right ventricle (tricuspid valve); from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve); and from the right ventricle to the main pulmonary (lung) artery (pulmonary valve)
- “Cardiomyopathy” is the medical term for disease of the heart muscle; “restrictive cardiomyopathy” is a disease in which the muscle is “stiff” and does not expand, such that blood cannot fill the ventricles normally
- “Restrictive cardiomyopathy” in cats is characterized by abnormal filling of the chambers of the heart (known as “diastolic dysfunction”), severe atrial enlargement, normal left ventricular wall thickness and variable abnormal pumping of the heart (known as “systolic dysfunction”)
- Scar tissue of the heart muscle layer may be present; in addition, other changes or damage in the muscle may be associated with other heart-muscle disorders, including inflammatory or immune-mediated diseases

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

SIGNS/OBSERVED CHANGES in the ANIMAL

If Cat Does Not Have Congestive Heart Failure (condition in which the heart cannot pump an adequate volume of blood to meet the body's needs)

- Some cats have no clinical signs
- Sluggishness (lethargy)
- Poor appetite and weight loss
- Fainting (known as “syncope”)—rare; usually indicates serious irregular heart beats (known as “arrhythmias”)
- Weakness or paralysis (signs of blockage of blood flow secondary to the presence of a blood clot in the artery [condition known as “arterial thromboembolism”])
- Depression
- Extreme weight loss with muscle wasting (known as “cachexia”)
- Rapid heart rate (known as “tachycardia”)
- Irregular heart beats (arrhythmias)
- Sequence of three heart sounds (known as a “gallop rhythm”), when listening to the heart with a stethoscope; heart beat sounds like a galloping horse instead of normal “lub-dub”
- May have a heart murmur

If Cat Has Congestive Heart Failure (condition in which the heart cannot pump an adequate volume of blood to meet the body's needs), cat has signs as previously described, plus the following:

- Difficulty breathing (known as “dyspnea”)
- Rapid breathing (known as “tachypnea”)
- Panting
- Open-mouth breathing
- Bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”)
- Abdominal swelling or distention
- Enlarged liver (known as “hepatomegaly”) or fluid build-up in the abdomen (known as “ascites”), with enlargement or distension of the jugular veins (located on either side of the neck)
- Short, rough snapping sounds (known as “crackles”) heard when listening to the chest with a stethoscope
- Muffled heart or lung sounds heard when listening to the chest with a stethoscope, if the cat has fluid build-up in the space between the chest wall and lungs (known as “pleural effusion”)
- Weakness or paralysis with loss of femoral pulses; one or more extremities may be cold and painful (signs of blockage of blood flow secondary to the presence of a blood clot in the artery [condition is “arterial thromboembolism”])

CAUSES AND RISK FACTORS

- True cause(s) unknown (so called “idiopathic disease”); often no “predisposing” disease can be documented
- Suspected initiating causes include inflammation of the heart muscle (known as “myocarditis”); inflammation of the inner muscle layer of the heart (known as “endomyocarditis”); infiltration of eosinophils (a type of white-blood cell) into the heart muscle (known as “eosinophilic myocardial infiltration”); disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle (known as “hypertrophic cardiomyopathy”) with sudden lack of blood supply to the heart muscle that leads to death of tissues (known as “myocardial infarction”); widespread (diffuse) “small blood vessel disease;” and other causes of abnormal blood flow and resulting lack of oxygen to the heart muscle



TREATMENT

HEALTH CARE

- Patients with sudden (acute), severe congestive heart failure are hospitalized for emergency care; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Animals that do not have clinical signs or have mild signs can be treated with outpatient medical management
- Animals with severe difficulty breathing (severe dyspnea) should receive oxygen via oxygen cage, nasal cannula, or mask (beware of stress to patient)
- Life-threatening fluid build-up in the space between the chest wall and lungs (pleural effusion) is reduced via tapping and draining the chest (known as “thoracocentesis”)
- Treat associated conditions (such as dehydration or low body temperature [hypothermia])
- Low sodium fluids administered cautiously if dehydration occurs (beware of worsening congestive heart failure)
- Heating pad may be necessary for patients with low body temperature (hypothermia)

ACTIVITY

- Maintain a low stress environment to decrease patient anxiety (such as cage rest, minimize handling)

DIET

- Low-salt diet may decrease fluid retention, but strict adherence to dietary changes should be avoided in sudden (acute) congestive heart failure in order to maintain food intake
- Hand feed, as necessary



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Sudden (Acute) Congestive Heart Failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)

- Medication to remove excess fluid from the body (diuretic) administered by injection—furosemide
- Dobutamine (drug to increase contraction of the heart muscle) to increase function of the heart
- Nitroglycerin ointment, applied to the skin
- Oxygen delivered by cage, mask, or nasal tube
- Tapping and draining the chest (thoracocentesis), as necessary to relieve difficulty breathing (dyspnea) due to fluid build-up in the space between the chest wall and lungs (pleural effusion)
- Severe irregular heart beats originating above the ventricles (supraventricular arrhythmias) may be treated with diltiazem
- Rapid ventricular heart rate (ventricular tachycardia) may resolve with resolution of congestive heart failure; treatment for sudden occurrence of ventricular tachycardia may include lidocaine
- Beta-blockers (such as propranolol or atenolol) may be used to treat irregular heart beats that originate above or in the ventricles (supraventricular or ventricular arrhythmias), but not until congestive heart failure is treated

Long-Term (Chronic) Therapy

- Medication to remove excess fluid from the body (diuretic)—furosemide, gradually decreased to lowest effective dose
- Long-term (chronic) therapy with diltiazem decreases heart rate and improves irregular heart beats that originate above the ventricles (supraventricular arrhythmias) and may improve heart function
- Beta-blockers may be used to slow heart rate and treat irregular heart beats that originate above or in the ventricles (supraventricular or ventricular arrhythmias)
- Angiotensin-converting enzyme (ACE) inhibitors may reduce fluid retention and decrease need for medications to remove excess fluid from the body (diuretics); examples of ACE inhibitors are enalapril and benazepril
- Digoxin (a heart medication) may be used if heart-muscle contraction is impaired or atrial fibrillation (rapid, irregular heart rhythm involving the top two chambers of the heart [atria]) is present
- Aspirin may be administered to prevent blood clots (known as “thromboembolism”), but effectiveness is questionable; administer aspirin only under the direction of your cat’s veterinarian
- Warfarin may be administered to prevent blood clots (thromboembolism), but is not recommended unless close monitoring and repeated measurement of prothrombin time (a blood test to evaluate clotting) are feasible



FOLLOW-UP CARE

PATIENT MONITORING

- Frequent serial physical examinations (minimal stress to patient) to assess response to treatment and resolution of fluid build-up in the lungs (pulmonary edema) and fluid build-up in body cavities (known as “effusions”)
- Frequent assessment of hydration and kidney function is important in first few days of therapy to avoid removal of too much fluid from the body (known as “over diuresis”) and development of excessive levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”)
- Repeated tapping and draining the chest (thoracocentesis) may be necessary to maintain the amount of fluid build-up in the space between the chest wall and lungs (pleural effusion) at a comfortable level
- “Hands-off” hourly assessment of breathing rate in first 12 to 24 hours can be used to monitor efficacy of congestive heart failure therapy
- Chest X-rays may be repeated in 12 to 24 hours
- Blood work (especially creatinine and potassium) should be monitored closely during the first 3 to 5 days of therapy to detect dehydration, kidney failure, and low levels of potassium in the blood (known as “hypokalemia”)—caused by medications to remove excess fluid from the body (diuretics); or high levels of potassium in the blood (known as “hyperkalemia”), if angiotensin-converting enzyme (ACE) inhibitors are administered
- Repeat physical examination and blood work (especially electrolyte analysis) after approximately 10 to 14 days of treatment
- Electrocardiograms (“ECGs,” recordings of the electrical activity of the heart) and X-rays may be repeated, as your cat’s veterinarian feels necessary
- Stable patients are reevaluated every 2 to 4 months, or more frequently if problems occur

POSSIBLE COMPLICATIONS

- Congestive heart failure
- Death

EXPECTED COURSE AND PROGNOSIS

- Highly variable, based on presentation of disease and clinical signs
- Most cats with restrictive cardiomyopathy and congestive heart failure live 3 to 12 months; some live 2 years



KEY POINTS

- “Restrictive cardiomyopathy” is a disease in which the heart muscle is “stiff” and does not expand, such that blood cannot fill the ventricles normally
- Patients with sudden (acute), severe congestive heart failure are hospitalized for emergency care; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Animals that do not have clinical signs or have mild signs can be treated with outpatient medical management
- Most cats with restrictive cardiomyopathy and congestive heart failure live 3 to 12 months; some live 2 years

CATARACTS



BASICS

OVERVIEW

- Opacity in the lens; the lens is the normally clear structure directly behind the iris (the colored part of the eye) that focuses light as it moves toward the back part of the eye (retina); if opacity is complete, it prevents passage of light to the back part of the eye (retina), leading to blindness in the affected eye
- “Cataract”—may refer to a lens that is entirely opaque or to a localized opacity within the lens; does not imply cause

GENETICS

- Most cataracts are inherited
- Most common mode of inheritance—simple autosomal recessive
- Some breeds—dominantly inherited

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Many dog breeds are affected by hereditary cataracts
- Cataracts that typically progress to blindness are found in miniature poodles; American cocker spaniels; miniature schnauzers
- Other commonly affected dog breeds—golden retrievers; Boston terriers; Siberian huskies
- Cats—Persians; Birmanians; Himalayans

Mean Age and Range

- Depend on cause
- Hereditary (dogs)—may be congenital (present at birth); may develop later in life (known as “acquired cataracts”) anytime from several months to many years of age, depending on the breed
- Hereditary (cats)—all reported to date have been congenital (present at birth)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Opacity of the lens
- Related to the degree of vision impairment
- Occupy less than 30% of the lens or affect only one eye—often go unnoticed
- Occupy more than 60% of the lens—usually noticed and reported to the veterinarian
- Cataract caused by diabetes mellitus (sugar diabetes)—may see signs of diabetes, such as increased urination (known as “polyuria”), increased thirst (known as “polydipsia”), and weight loss
- Cloudiness in the eye (specifically the lens) noticed before vision impairment—usually related to sclerosis, rather than cataract formation; “sclerosis” is a normal aging change in the lens due to changes in the lens fibers, it apparently has little to no effect on vision
- Associated deterioration of the back of the eye (retina; condition known as “progressive retinal degeneration”) in dogs—difficulty seeing in dimly lighted conditions (known as “nyctalopia” or “night blindness”)
- May be associated with inflammation of the front part of the eye, including the iris (known as “anterior uveitis”)—typically see cloudiness of aqueous humor (the “aqueous humor” is the transparent liquid that fills the front part of the eyeball) due to increased protein content and suspended cellular debris (condition known as “aqueous flare”); scar tissue between the iris and the lens of the eye (known as “synechiae”); and decreased pressure within the eye (known as “low intraocular pressure”)

CAUSES

- Heredity
- Diabetes mellitus (sugar diabetes)
- Spontaneous—age-related
- Advanced deterioration of the back of the eye (retinal degeneration)—response to toxic dialdehydes (type of chemical, used in disinfectants and leather tanning products)
- Inflammation of the front part of the eye, including the iris (anterior uveitis)—secondary to formation of scar tissue between the iris and the lens of the eye (synechia) or altered aqueous humor (the transparent liquid that fills the front part of the eyeball) composition
- Toxic substances—dinitrophenol; naphthalene
- Nutrition—milk-replacer diet
- Low levels of calcium in the blood (known as “hypocalcemia”)
- Radiation
- Electric shock

RISK FACTORS

- Genetics
- Multiple congenital (present at birth) eye defects

- Any disease capable of causing inflammation of the front of the eye, including the iris (anterior uveitis)
- Advanced deterioration of the back of the eye (retinal degeneration)
- Generalized (systemic) metabolic diseases—diabetes; diseases capable of causing low levels of calcium in the blood (hypocalcemia)



TREATMENT

HEALTH CARE

- Dogs undergoing cataract surgery—inpatient or outpatient
- Hospitalization—rarely required for more than 48 hours

SURGERY

- Phacoemulsification is a surgical procedure in which ultrasonic vibrations are used to fragment and liquefy the lens, in order to remove the lens material; procedure of choice
- Prognosis for successful surgery—generally greater than 90%; depends on the stage of the cataract and other possible abnormalities in the eye
- Intraocular lenses—may be implanted safely at the time of surgery, so patient will not suffer extreme farsightedness



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Prednisolone acetate (1%) to prevent and control lens-induced inflammation of the front part of the eye, including the iris (anterior uveitis)
- OcluVet™ Eye Drops recently have been marketed with claims that topical application (that is, applied to the eye) reduces lens opacification in dogs; preliminary reports from veterinary eye doctors (ophthalmologists) do not support the claim of effectiveness—further studies need to be performed



FOLLOW-UP CARE

PATIENT MONITORING

- All patients—monitor carefully for progression of cataracts
- Hereditary—cataracts may progress very quickly in young dogs

PREVENTIONS AND AVOIDANCE

- Do not breed patients with known or suspected inherited conditions

POSSIBLE COMPLICATIONS

- Complete cataracts—potential to cause lens-induced inflammation of the front part of the eye, including the iris (anterior uveitis); secondary glaucoma (in which the pressure within the eye [intraocular pressure] is increased secondary to inflammation in the front part of the eye); and the separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid;” condition known as “retinal detachment”)

EXPECTED COURSE AND PROGNOSIS

- Rate of progression—depends on location of cataract within the lens and the patient’s age
- Diabetes mellitus—induced cataract—usually very rapid progression
- Surgical intervention—for hereditary or diabetes-caused cataracts, prognosis for good vision excellent; for other types of cataracts, depends on cause



KEY POINTS

- Surgery normally can be done on any hereditary cataract that is causing or is anticipated to cause vision loss

- Prognosis for surgery is better if it is done early in the course of cataract development, before the cataract changes to the point where the lens is smaller and may actually “clear” to some degree (known as “hypermaturation” of the cataract), lens-induced inflammation of the front part of the eye, including the iris (anterior uveitis), and/or separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment) occur
- It is not advisable to delay surgery until the patient is blind in both eyes
- Surgery may or may not be indicated for nonhereditary cataracts; discuss surgery with your pet’s veterinarian
- With the high success rate of phacoemulsification (surgical procedure in which ultrasonic vibrations are used to fragment and liquefy the lens, in order to remove the lens material), it is no longer appropriate to observe cataracts for possible resorption, even in young dogs

LUMBOSACRAL STENOSIS AND CAUDA EQUINA SYNDROME



BASICS

OVERVIEW

- Pressure to or damage of the nerves within the spinal canal in the area of the junction between the lumbar and sacral vertebrae; at this level of the spine, spinal nerves are located in the spinal canal (rather than spinal cord)—these spinal nerves within the spinal canal are known as the “cauda equina”
- Caused by narrowing of the lumbosacral spinal canal with compression of the seventh lumbar (L_7), sacral, or caudal nerve roots
- Syndrome refers to the clinical signs related to injury of these nerve roots
- The narrowing of the lumbosacral spinal canal can be a congenital condition, which is present at birth, or an acquired condition, which develops sometime later in life/after birth
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C_1 - C_7 ; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T_1 - T_{13} ; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L_1 - L_7 ; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae
- Each disk is composed of a central gel-like area, known as the “nucleus pulposus,” and an outer fibrous ring, known as the “annulus fibrosis”
- Degeneration of the intervertebral disks causes protrusion of disk material into the spinal canal; the protruded disk material causes pressure on the spinal cord
- Protrusion is defined as the disk bulging into the spinal canal with the fibrous ring of the disk being intact
- Two types of protrusion have been reported in dogs: sudden (acute) disk herniation (“slipped disk”) is Hansen type I and long-term (chronic) disk herniation is Hansen type II; Hansen type II involves degeneration of the disk, followed by bulging of the disk into the spinal cord with the fibrous ring remaining intact (protrusion)

GENETICS

- No known genetic basis

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Common in dogs
- Rare in cats

Breed Predilections

- Congenital (present at birth)—small to medium dogs; border collies
- Acquired (condition that develops sometime later in life/after birth)—large-breed dogs; German shepherd dogs, boxers, rottweilers

Mean Age and Range

- Congenital (present at birth)—signs seen at 3 to 8 years of age
- Acquired (condition that develops sometime later in life/after birth)—average age at onset of signs is 6 to 7 years

Predominant Sex

- Congenital (present at birth)—none
- Acquired (condition that develops sometime later in life/after birth)—male

SIGNS/OBSERVED CHANGES in the ANIMAL

- Relate to varying degrees of compression of the seventh lumbar (L_7), sacral, and caudal nerve roots
- Lumbosacral pain—salient clinical feature; may be the only sign
- Sciatic nerve dysfunction—the sciatic nerve is the largest nerve in the body; it travels from the pelvis/hip down the thigh; dysfunction initially may cause lameness; may progress to rear leg weakness, muscle wasting, and abnormal reflexes
- Pudendal nerve-root involvement—the pudendal nerve starts from the sacral nerves and provides innervation to the area of the rectum, external genitalia, and perineum (the skin between the anus and external genitalia); pudendal nerve-root involvement may lead to inability to control urination (known as “urinary incontinence”) and/or inability to control bowel movements (known as “fecal incontinence”)
- Caudal nerve-root involvement—abnormal tail carriage; weakness to paralysis of the tail

- Both meninges (the membranes covering the spinal cord) and nerve-root compression—sensory disturbances that vary from unpleasant sensations to obvious low lumbar pain
- Extension of the rear legs or movement of the tail over the back reduces the lumbosacral spinal canal diameter and usually elicits a painful response
- Congenital (present at birth)—self-inflicted lesions secondary to pain are common

CAUSES

- Congenital (present at birth) malformation of the backbones (vertebrae), including transitional vertebra (abnormal development of the backbone at the junction between two vertebral types; in this case, at the junction of the lumbar and sacral vertebrae) or osteochondrosis of the sacral endplates; “osteochondrosis” is a disorder of bone formation in the growth plates (areas where bone grows in length in the young animal), in this case, involving the sacral backbones (vertebrae)
- Hansen type II disk protrusion
- Increase in size (known as “hypertrophy” or “hyperplasia”) of the inter-arcuate ligaments (ligaments located between adjacent backbones [vertebrae])
- Proliferation of the articular facets (surfaces of the backbone [vertebra] where it joins together with another back bone)
- Partial dislocation (known as “subluxation”) or instability of the area of the junction between the lumbar and sacral backbones (vertebrae)

RISK FACTORS

- Dogs, especially German shepherd dogs, with a lumbosacral transitional vertebra (abnormal development of the backbone at the junction between two vertebral types; in this case, at the junction of the lumbar and sacral vertebrae) have increased risk to develop the syndrome



TREATMENT

HEALTH CARE

- Lack of control of urination (urinary continence)—outpatient, pending surgery
- Lack of control of urination (urinary incontinence)—inpatient for initial medical management
- Lack of control of urination (urinary incontinence)—catheterize the bladder until adequate voluntary control of urination returns; monitor closely for urinary tract infection and administer appropriate antibiotics, if necessary

ACTIVITY

- After surgery—restrict for 4 weeks; then gradually return to athletic function
- Nonsurgical treatment—confinement and restricted leash walks, alone or combined with steroids, frequently alleviates pain; clinical signs often return with increasing levels of exercise

DIET

- Avoid obesity; excess weight increases stress on the spine

SURGERY

- Surgery to relieve pressure on the nerves (known as “decompression”)—preferred treatment; various surgical procedures may be performed



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs) or steroids—used to decrease inflammation; usually unsatisfactory
- Lack of control of urination (urinary incontinence)—administer appropriate antibiotics, if patient develops urinary tract infection



FOLLOW-UP CARE

PATIENT MONITORING

- Evaluate nervous system status
- Lack of control of urination (urinary incontinence)—monitor closely for urinary tract infection

POSSIBLE COMPLICATIONS

- Accumulation of fluid (serum) in the tissues, causing the development of a mass (known as “seroma formation”)—frequent sequela to surgery; can be managed effectively by cage rest and surgical drainage of the fluid
- Excessive scar formation in the surgical area—infrequent cause of recurrence of clinical signs
- Recurrence of signs more than 6 months after surgery

EXPECTED COURSE AND PROGNOSIS

- Vary with degree of nerve injury
- Low lumbar pain and mild nervous system deficits (dogs)—good prognosis after surgery; 70% to 80% have an excellent or good outcome
- Inability to control urination (urinary incontinence) or bowel movements (fecal incontinence) in dogs—guarded prognosis



KEY POINTS

- Without treatment, the pet will have progressive nervous system impairment of the rear legs, lack of control of urination (urinary incontinence) and bowel movements (fecal incontinence), and paralysis of the tail
- Rear-leg lameness and self-inflicted lesions result from pain associated with nerve-root irritation and compression
- Discuss surgical treatment with your pet’s veterinarian; the goal of surgery is to stop the progression of nervous system impairment and to remove the source of pain
- Some nervous system deficits may remain following surgery
- Medical management alone usually is unsatisfactory

DISEASE CAUSED BY *CHLAMYDOPHILA*, A TYPE OF BACTERIA (CHLAMYDIOSIS) IN CATS



BASICS

OVERVIEW

- A long-term (chronic) respiratory tract infection of cats caused by an intracellular bacteria, *Chlamydophila felis*
- Characterized by inflammation of the moist tissues of the eye (known as “conjunctivitis”), mild upper respiratory signs, and mild inflammation of the lungs (known as “pneumonitis”)
- The respiratory tract consists of the “upper respiratory tract” (the nose, nasal passages, throat, and windpipe [trachea]) and the “lower respiratory tract” (the bronchi, bronchioles, and alveoli [the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged])

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats
- People

Mean Age and Range

- Usually kittens 2 to 6 months of age; any age cat possible

SIGNS/OBSERVED CHANGES in the ANIMAL

- Cat may be infected, but have no signs of disease (known as a “subclinical infection”)
- Clinical disease only develops if the animal has a simultaneous infection by other disease-causing agents (such as viruses)
- Upper respiratory infection, with some sneezing, watery eyes, and coughing
- Sometimes difficult breathing (known as “dyspnea”)
- Varying degree of lack of appetite (known as “anorexia”)
- Inflammation of the moist tissues of the eyes (conjunctivitis)—often granular; initially involving one eye, sometimes involving both eyes
- Excessive production of tears (known as “lacrimation”); avoidance of light (known as “photophobia”); and squinting or spasmodic blinking (known as “blepharospasm”)
- Inflammation of the nose (known as “rhinitis”) with discharge from the nose—usually mild
- Inflammation of the lungs (pneumonitis)—with the inflammatory process in the alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged), bronchioles and airways producing abnormal breath sounds as heard when listening to the chest with a stethoscope (known as “auscultation”)

CAUSES

- *Chlamydophila felis*
- *Chlamydia* species

RISK FACTORS

- Simultaneous infections with other respiratory disease-causing agents (such as viruses)
- Lack of vaccination
- Multi-cat facilities, especially adoption shelters and breeding catteries



TREATMENT

HEALTH CARE

- Generally as an outpatient
- Keep nostrils and eyes clean of discharge
- Generally does not require other supportive therapy (such as administration of fluids), unless complicated by simultaneous infections

ACTIVITY

- Quarantine affected cats from contact with other cats
- Do not allow affected cats to go outside

DIET

- Normal



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics administered by mouth (systemic treatment)—tetracycline; doxycycline
- Antibiotics applied to the eye directly (topical treatment)—eye ointments containing tetracycline
- Other antibiotics are generally less effective than tetracycline



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor for improved health as treatment proceeds

PREVENTIONS AND AVOIDANCE

- Vaccines are available to reduce the severity and duration of infection; vaccines do not prevent infection
- Discuss the need for vaccinating your cat against this disease with the veterinarian

POSSIBLE COMPLICATIONS

- Adverse vaccine reactions—mild clinical disease following administration of modified live vaccines; occurs in a small percentage of vaccinated cats

EXPECTED COURSE AND PROGNOSIS

- Tends to be long-term (chronic) disease, lasting for several weeks or months, unless successful antibiotic treatment is given
- Prognosis good



KEY POINTS

- Vaccines are available to reduce the severity and duration of infection; vaccines do not prevent infection
- Tends to be long-term (chronic) disease, lasting for several weeks or months, unless successful antibiotic treatment is given
- Clinical disease only develops if the animal has a simultaneous infection by other disease-causing agents (such as viruses)

CHOCOLATE POISONING



BASICS

OVERVIEW

- Sudden (acute) gastrointestinal, nervous system, and heart problems caused by excessive intake of chemicals (known as methylxanthine alkaloids, such as theobromine and caffeine) present in chocolate

SIGNALMENT/DESCRIPTION of ANIMAL

- Dogs and rarely cats
- Small dogs—may be more at risk (amount of chocolate available compared to dog's body weight)
- Puppies and young dogs—may be more likely to ingest large amounts of unusual foods

SIGNS/OBSERVED CHANGES in the ANIMAL

Signs are seen after recent chocolate ingestion

- Vomiting and diarrhea—often the first reported signs; occur 2–4 hours after ingestion
- Early restlessness and increased activity
- Frequent urination (polyuria)—may result from diuretic action of chemicals in chocolate
- Advanced signs—stiffness; excitement; seizures

CAUSES

- Usually some form of processed chocolate (used for baking and candies, such as milk chocolate)—contain high concentrations of theobromine and caffeine

RISK FACTORS

- Dogs—most commonly affected because they consume large amounts of unusual foods quickly
- Chocolate—highly palatable or tasty and attractive; often readily available and unprotected in homes and kitchens, especially around the holidays when chocolate products and candies are common



TREATMENT

HEALTH CARE

- Describe the type of chocolate and amount of exposure to your veterinarian; take your pet to a veterinary hospital as a potential poisoning emergency
- Fluid therapy—correct electrolyte disturbances caused by vomiting, as necessary
- Control seizures
- Detoxification (if not having seizures or seizures are controlled)—your veterinarian may induce vomiting, flush the stomach with fluids (gastric lavage), and administer activated charcoal
- Control overheating (hyperthermia)
- Treat rapid heart rate (tachycardia)

ACTIVITY

- Avoid stress and excitement as these could make nervous system signs (hyperreflexia or seizures) worse

DIET

- Acutely affected patient—do not feed
- Recovering or convalescent patient—bland diet for several days to allow recovery from gastrointestinal problems



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Induce vomiting (emesis)—*only if patient is not having seizures*—apomorphine, syrup of ipecac, or 3% hydrogen peroxide

- Flush the stomach (gastric lavage)—only before onset of vomiting and other clinical signs or vomiting has been controlled, if drugs to induce vomiting are not effective, seizures are controlled, and an endotracheal tube is in place
- Once vomiting is controlled—activated charcoal to attract and keep the remaining alkaloids in the gastrointestinal tract
- Osmotic cathartic—sodium sulfate or sorbitol 70% promotes gastrointestinal elimination of chocolate
- Hyperactivity and seizures—controlled with diazepam
- Ventricular rapid heart rate (known as “tachycardia”) in dogs—lidocaine (without epinephrine); lidocaine is not recommended in cats
- Serious abnormal heart rhythms (arrhythmias) that persist after medical treatment—metoprolol or propranolol; metoprolol preferred but may be difficult to obtain; may use oral therapy once patient is stable; monitor electrocardiogram (ECG) and watch for hypotension (a possible complication to this treatment)
- Control may be obtained with methacarbamol
- If response to diazepam inadequate—consider phenobarbital
- For refractory seizures—pentobarbital



FOLLOW-UP CARE

PATIENT MONITORING

- Electrocardiogram (ECG) to evaluate and monitor abnormal heart rhythms (arrhythmias)
- Watch for mild to moderate kidney disease (nephrosis) in recovering patients

PREVENTIONS AND AVOIDANCE

- Chocolate is among the 20 most common poisonings reported in recent literature, by the National Animal Poison Control Center, and by the Hennepin County (Minneapolis) Poison Control Center
- Keep chocolate in a secure location, out of reach of pets

POSSIBLE COMPLICATIONS

- Pregnant animal—risk for abnormal development of fetus leading to birth defects of newborns
- Nursing animals—over stimulation of nervous system in nursing newborns

EXPECTED COURSE AND PROGNOSIS

- Expected course—12–36 hours, depending on dosage of chocolate and effectiveness of decontamination and treatment
- Successfully treated patients—usually recover completely
- Prognosis—good if oral decontamination occurs within 2–4 hours of ingestion; guarded with advanced signs of seizures and arrhythmias



KEY POINTS

- Chocolate ingestion is hazardous to pets; if you suspect your pet has eaten chocolate, contact your veterinarian immediately
- Describe the type of chocolate and amount of exposure to your veterinarian; take your pet to a veterinary hospital as a potential poisoning emergency
- Chocolate is among the 20 most common poisonings reported in recent literature by the National Animal Poison Control Center and by the Hennepin County (Minneapolis) Poison Control Center
- Keep chocolate in a secure location, out of reach of pets
- Be especially careful around holidays when chocolate products and candies are readily available

CHOLANGITIS/CHOLANGIOHEPATITIS SYNDROME

(INFLAMMATION OF THE BILE DUCT SYSTEM AND LIVER)



BASICS

OVERVIEW

- The liver is the largest gland in the body; it has many functions, including production of bile (a fluid substance involved in digestion of fats); bile ducts begin within the liver itself as tiny channels to transport bile—the ducts join together to form larger bile ducts and finally enter the extrahepatic or common bile duct, which empties into the upper small intestine; the system of bile ducts is known as the “biliary tree”
- The gallbladder is the storage unit for bile; bile is stored until it is needed for fat digestion
- “Cholangitis” is inflammation of the bile duct or the biliary tree
- “Cholangiohepatitis” is inflammation of biliary structures and surrounding liver tissue
- Cholangitis/cholangiohepatitis syndrome occurs primarily in cats; it is classified as “suppurative” or “nonsuppurative” (lymphoplasmacytic, lymphocytic), “granulomatous,” or “lymphoproliferative” (transition to lymphoma) based on microscopic examination of biopsy samples
- “Suppurative” refers to the presence of pus in the affected tissue; “nonsuppurative” refers to an inflammatory process that is not characterized by the presence of pus—in cholangitis/cholangiohepatitis syndrome, the inflammatory process is characterized by the presence of lymphocytes and plasma cells (so called “lymphoplasmacytic” disease) or lymphocytes (so called “lymphocytic” disease); lymphocytes are a type of white-blood cell that are formed in lymphatic tissues throughout the body—lymphocytes are involved in the immune process; plasma cells are specialized white-blood cells; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease
- “Granulomatous” refers to nodular, inflammatory lesions; “lymphoproliferative” refers to conditions in which an excessive number of lymphocytes are produced; “lymphoma” is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body

SIGNALMENT/DESCRIPTION OF ANIMAL

Species

- Cats (common) and dogs (uncommon)

Breed Predispositions

- Cats—possibly Himalayan, Persian, and Siamese

Mean Age and Range

- Suppurative cholangitis/cholangiohepatitis syndrome—range, 0.4 to 16 years of age; mostly young to middle-aged cats
- Nonsuppurative cholangitis/cholangiohepatitis syndrome—range, 2 to 17 years of age; mostly middle-aged cats

Predominant Sex

- Suppurative cholangitis/cholangiohepatitis syndrome—male cats more likely to be affected than female cats
- Nonsuppurative cholangitis/cholangiohepatitis syndrome—none

SIGNS/OBSERVED CHANGES in the ANIMAL

- Suppurative cholangitis/cholangiohepatitis syndrome—most severe clinical illness characterized by “acute abdomen” (sudden onset of severe abdominal pain), fever, often less than 5 days’ duration; associated with blockage of the extrahepatic or common bile duct (known as “extrahepatic bile duct obstruction”)
- Suppurative cholangitis/cholangiohepatitis syndrome—sudden (acute) illness; fever; lack of appetite (known as “anorexia”); vomiting; painful abdomen; may have yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”); dehydration; collapse; shock
- Nonsuppurative cholangitis/cholangiohepatitis syndrome—illness of greater than 3 weeks’ duration (may have signs of illness for months to years); cyclic illness; long-term (chronic) vague signs: sluggishness (lethargy), vomiting, lack of appetite (anorexia), and weight loss; few physical abnormalities other than enlarged liver (known as “hepatomegaly”); thickened intestines with inflammatory bowel disease (IBD); variable yellowish discoloration to the gums and other tissues of the body (jaundice or icterus); rare fluid build-up in the abdomen (known as “abdominal effusion” or “ascites”)
- Decreased number of bile ducts (known as “ductopenia”) associated with nonsuppurative cholangitis/cholangiohepatitis syndrome in cats—increased appetite (known as “polyphagia”) due to reduced bile flow with poor digestion of fats and presence of large amounts of fat in the stool, due to the inability to digest the fat (known as “steatorrhea”) leading to decreased levels of fat-soluble substances (such as vitamin K₁, essential fatty acids, vitamin E); unkempt coat, variable hair loss on the sides of the chest; variable pale or grayish coloration to the stools (known as “acholic feces”), due to the lack of bile pigments that cause the normal brown color of bowel movements

CAUSES

Suppurative Cholangitis/Cholangiohepatitis Syndrome

- Bacterial infection—most common in cats: *E. coli*, *Enterobacter*, *Enterococcus*, β -hemolytic *Streptococcus*, *Klebsiella*, *Actinomyces*, *Clostridia*, and *Bacteroides*; also rarely associated with toxoplasmosis; dogs: intestinal bacterial opportunists (bacteria that usually do not

cause disease, but are able to cause disease because the animal's body and/or immune system has been weakened by some other disease process); *Campylobacter*, *Salmonella*, and *Leptospira*

- Common condition following blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction) and diseases in which flow of bile is decreased or stopped (known as "cholestasis")

Nonsuppurative Cholangitis/Cholangiohepatitis Syndrome

- Coexistent disorders— inflammation of the gallbladder (known as "cholecystitis"); presence of hard, solid material in the bile duct or gallbladder (known as "cholelithiasis"); inflammation of the pancreas (known as "pancreatitis"); blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction); inflammatory bowel disease (IBD); long-term (chronic) inflammation of the tissue spaces in the kidneys (known as "chronic interstitial nephritis")

RISK FACTORS

- Suppurative cholangitis/cholangiohepatitis syndrome—blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction); diseases in which flow of bile is decreased or stopped (cholestasis); infections elsewhere in the body
- Nonsuppurative cholangitis/cholangiohepatitis syndrome—inflammatory bowel disease (IBD); inflammation of the pancreas (pancreatitis); blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction); possibly long-term (chronic) inflammation of the tissue spaces in the kidneys (chronic interstitial nephritis)



TREATMENT

HEALTH CARE

Inpatient Management

- Suppurative cholangitis/cholangiohepatitis syndrome with sudden (acute) illness characterized by fever, painful abdomen, abnormal white-blood cell count—hydration support; antibiotics; if patient has blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction) or inflammation of the gallbladder (cholecystitis): administer antibiotics *before* surgery; continue antibiotics for at least 8 weeks; treat with medications (such as ursodeoxycholic acid, SAME) to improve the secretion of bile until liver enzyme values normalize on blood work (serum biochemistry profile)
- Nonsuppurative cholangitis/cholangiohepatitis syndrome in cats with clinical signs of disease—fluid therapy, as necessary; diagnostic evaluations; liver biopsy (administer vitamin K₁ *before* liver biopsy)
- Both forms of cholangitis/cholangiohepatitis syndrome in cats—may need blood transfusion following surgery or biopsy
- Supplement fluids with B vitamins, potassium chloride, and potassium phosphate, as needed; avoid dextrose supplements

Outpatient Management

- Suppurative cholangitis/cholangiohepatitis syndrome—after sudden (acute) crisis has been managed
- Nonsuppurative cholangitis/cholangiohepatitis syndrome—after resolution of sudden (acute) crisis, provide lifelong treatment to alter the immune system (known as "immune-system modulation") as well as antioxidant and liver-protective therapy

ACTIVITY

- Restricted while patient has clinical signs of disease

DIET

- Nutritional support—to avoid hepatic lipidosis, feed a balanced high-protein, high-calorie feline diet; supplement water-soluble vitamins (such as vitamin B); "hepatic lipidosis" is a disease in which fats and lipids (compounds that contain fats or oils) accumulate in the liver as a possible complication of lack of appetite (anorexia)
- Antigen-restricted diet with coexistent inflammatory bowel disease (IBD)
- Fat-restricted diet, if patient has severe decrease in bile ducts (ductopenia), abnormal absorption of fat (known as "fat malabsorption"), or long-term (chronic) inflammation of the pancreas (pancreatitis) causing abnormal digestion of food (known as "maldigestion")
- May require feeding tubes; rarely requires feeding through intravenous fluids (known as "parenteral nutrition")

SURGERY

- Surgical removal of the gallbladder (known as "cholecystectomy")—if the patient has inflammation of the gallbladder (cholecystitis)
- Surgical procedure to produce a new connection between the biliary tree and the small intestines (known as "cholecystoenterostomy")—may be needed in patients with blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction)
- Surgical removal of stones in the gallbladder (stones known as "choleliths")



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antibiotics for Suppurative Cholangitis/Cholangiohepatitis Syndrome

- Antibiotics that kill bacteria (known as “bactericidal antibiotics”)—against intestinal bacterial opportunists (bacteria that usually do not cause disease, but are able to cause disease because the animal’s body and/or immune system has been weakened by some other disease process); amoxicillin-clavulanic acid (Clavamox®) or enrofloxacin combined with metronidazole
- Resistant enterococci—vancomycin
- Modify antibiotics, based on bacterial culture and sensitivity reports

Medications to Alter the Immune Response (Immunomodulation) for Nonsuppurative Cholangitis/Cholangiohepatitis Syndrome

- Steroids—prednisolone; long-term (chronic) therapy usually needed
- Metronidazole—in combination with prednisolone, especially if patient has coexistent inflammatory bowel disease (IBD)
- Cats with confirmed decreased number of bile ducts (ductopenia) require more aggressive treatment; clinical experience suggests combination of prednisolone, metronidazole with pulsed methotrexate (a chemotherapeutic drug); provide folate (folinic acid)
- Some cats require chemotherapy protocols developed for lymphoma (type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body)

Antioxidants

- Vitamin E (tocopherol)—higher dose if patient has long-term (chronic) blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction) or decrease in the number of bile ducts (ductopenia) because of abnormal absorption of fat (fat malabsorption)
- S-adenosylmethionine (SAmE, Denosyl® SD4)

Other

- Ursodeoxycholic acid—has numerous potentially beneficial effects, including altering the immune response (immunomodulation), protecting the liver, causing secretion of bile, and providing antioxidant effects
- B-vitamin supplementation with thiamine (B₁) and B₁₂



FOLLOW-UP CARE

PATIENT MONITORING

- Nonsuppurative cholangitis/cholangiohepatitis syndrome—initially, monitor blood work (liver enzyme and bilirubin levels) every 7 to 14 days; with remission, assess blood work quarterly

PREVENTIONS AND AVOIDANCE

- Control inflammatory bowel disease (IBD)

POSSIBLE COMPLICATIONS

- Suppurative cholangitis/cholangiohepatitis syndrome may transform into nonsuppurative cholangitis/cholangiohepatitis syndrome or sclerosing cholangitis/cholangiohepatitis syndrome (characterized by thickening or hardening of the biliary and/or liver tissues)
- Diabetes mellitus in 30% of cats with sclerosing cholangitis/cholangiohepatitis syndrome treated with prednisolone
- Hepatic lipidosis with inadequate nutritional intake; “hepatic lipidosis” is a disease in which fats and lipids (compounds that contain fats or oils) accumulate in the liver of cats

EXPECTED COURSE AND PROGNOSIS

- Suppurative cholangitis/cholangiohepatitis syndrome—may be cured
- Nonsuppurative cholangitis/cholangiohepatitis syndrome—long-term (chronic) disease; long-term remission possible (remission greater than 8 years has been documented)



KEY POINTS

- “Cholangitis” is inflammation of the bile duct or the biliary tree
- “Cholangiohepatitis” is inflammation of biliary structures and surrounding liver tissue
- Cholangitis/cholangiohepatitis syndrome occurs primarily in cats; it is classified as “suppurative” or “nonsuppurative” (lymphoplasmacytic, lymphocytic), “granulomatous,” or “lymphoproliferative” based on microscopic examination of biopsy samples
- Suppurative cholangitis/cholangiohepatitis syndrome—sudden (acute) illness; fever; lack of appetite (anorexia); vomiting; painful abdomen; may have yellowish discoloration to the gums and other tissues of the body (jaundice or icterus); dehydration; collapse; shock
- Nonsuppurative cholangitis/cholangiohepatitis syndrome—illness of greater than 3 weeks’ duration (may have signs of illness for months to years); cyclic illness; long-term (chronic) vague signs: sluggishness (lethargy), vomiting, lack of appetite (anorexia), and weight loss; few physical abnormalities other than enlarged liver (hepatomegaly); thickened intestines with inflammatory bowel disease (IBD); variable yellowish discoloration to the gums and other tissues of the body (jaundice or icterus); rare fluid build-up in the abdomen (ascites)
- Long-term (chronic) nature of nonsuppurative cholangitis/cholangiohepatitis syndrome requires lifelong therapy

INFLAMMATION OF THE BACK PART OF THE EYE (CHORIORETINITIS)



BASICS

OVERVIEW

- Inflammation of the choroid and retina; the choroid is located immediately under the retina and is part of the middle-layer of the eyeball that contains the blood vessels; the retina contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision
- Choroid is also called “posterior uvea;” the uvea is the entire middle layer of the eyeball that contains the blood vessels; it is composed of the iris (the colored or pigmented part of the eye), the ciliary body (the area between the iris and the choroid), and the choroid (located under the retina)
- Diffuse inflammation may result in frank separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid;” condition known as “retinal detachment”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Generalized (systemic) fungal infections (known as “mycoses”)—more common in large, hunting-breed dogs
- Uveodermatologic syndrome—a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris (known as “anterior uveitis”), inflammation of the posterior uvea or choroid (known as “posterior uveitis”), or both and coexistent inflammation of the skin (known as “dermatitis”), characterized by loss of pigment in the skin of the nose and lips; Akitas, chows, and Siberian huskies are more likely to develop syndrome than other breeds
- Borzoi breed-specific eye disorder with multiple areas of fluid build-up in the retina (known as “retinal edema”) or loss of tissue in the choroid and retina (known as “chorioretinal atrophy”) resulting in deterioration of the back of the eye (retina), causing pigmented and hyper-reflective areas (known as “Borzoi chorioretinopathy”)

Mean Age and Range

- Depend on underlying cause

Predominant Sex

- Uveodermatologic syndrome—a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris (anterior uveitis), inflammation of the posterior uvea or choroid (posterior uveitis), or both and coexistent inflammation of the skin (dermatitis), characterized by loss of pigment in the skin of the nose and lips; more common in young male dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Not usually painful, except when the front part of the eye, including the iris (anterior uvea) is affected
- Vitreous abnormalities—the “vitreous” is the clear, gel-like material that fills the back part of the eyeball (between the lens and the retina); may note inflammatory substances (known as “exudates”), bleeding (hemorrhage), or evidence of the gel becoming liquified (known as “syneresis”)
- Interruption or change of course of the blood vessels in the back of the eye (retina) due to changes in the contour/surface of the retina
- Invasion of the eye by fly larvae (known as “ophthalmomyiasis”); usually seen in cats—tracts from migrating larvae may be seen when the eye is examined with an ophthalmoscope
- Changes in the appearance of the retina when examined with an ophthalmoscope; may include change in color, darkened or lighter areas, and scars
- Other signs related to underlying disease
- Few or small lesions—may note no apparent visual deficits

CAUSES

Dogs

- Viral infection—canine distemper virus; herpesvirus (rare, usually seen in newborn puppies); rabies virus
- Bacterial or rickettsial infections—generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”) or bacteria in the blood (known as “bacteremia”); leptospirosis; brucellosis; inflammation with accumulation of pus in the uterus (known as “pyometra”) that leads to toxic inflammation of the uvea (uveitis); *Borrelia* (Lyme disease); ehrlichiosis; Rocky Mountain spotted fever; bartonellosis
- Fungal or mycotic infection—aspergillosis; blastomycosis; coccidioidomycosis; histoplasmosis; cryptococcosis
- Algal infections—geotrichosis; protothecosis
- Parasitic—migration of parasitic larvae through the eye (known as “ocular larval migrans;” parasites include *Strongyles*, *Ascarids*, *Baylisascaris*); toxoplasmosis; leishmaniasis; *Neospora*; invasion of the eye by fly larvae (ophthalmomyiasis)
- Autoimmune disease—diseases in which the immune system attacks the body’s own tissues; examples include uveodermatologic syndrome (a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris [anterior uveitis],

inflammation of the posterior uvea or choroid [posterior uveitis], or both and coexistent inflammation of the skin [dermatitis], characterized by loss of pigment in the skin of the nose and lips) and systemic lupus erythematosus (autoimmune disease in which body attacks its own skin and other organs)

- Unknown cause (so called “idiopathic disease”)—Borzoï chorioretinopathy is an acquired syndrome where affected dogs have multiple areas of fluid build-up in the retina (known as “retinal edema”) or loss of tissue in the choroid and retina (known as “chorioretinal atrophy”); possibly genetic, but may be caused by some type of infection

Cats

- Viral infection—feline leukemia virus (FeLV); feline immunodeficiency virus (FIV); feline infectious peritonitis (FIP)
- Bacterial infection—generalized disease caused by the spread of bacteria in the blood (septicemia or blood poisoning) or bacteria in the blood (bacteremia); bartonellosis
- Fungal or mycotic infection—cryptococcosis; histoplasmosis; blastomycosis; others
- Parasitic—toxoplasmosis; invasion of the eye by fly larvae (ophthalmomyiasis)—fly larvae include Diptera, *Cuterebra*; migration of parasitic larvae through the eye (known as “ocular larval migrans”); leishmaniasis (one report)
- Protozoal infection—toxoplasmosis
- Autoimmune disease—diseases in which the immune system attacks the body’s own tissues; examples include periarteritis nodosa and systemic lupus erythematosus (autoimmune disease in which body attacks its own skin and other organs)

Dogs and Cats

- Infection introduced by some external event—wound that enters the eyeball or migrating foreign body; surgery that enters the eyeball (known as “intraocular surgery”)
- Infection spread through the blood or body tissues to the eyeball—generalized (systemic) disease spreading into the eye; may extend from the central nervous system via the nerve between the brain and the eye (the optic nerve)
- Metabolic—early lesions in the back part of the eye (retina) secondary to high blood pressure (known as “hypertensive retinopathy lesions”) may appear as multiple areas of inflammation of the retina (known as “multifocal retinitis”)
- Generalized disease caused by the spread of bacteria in the blood (septicemia or blood poisoning) or bacteria in the blood (bacteremia) — bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition known as “diskospondylitis”); inflammation/infection of the lining of the heart (known as “endocarditis”); inflammation with accumulation of pus in the uterus (known as “pyometra”); may result from primary infection or associated immune-complex disease
- Cancer—primary cancer involving the choroid and/or retina or secondary to the spread of cancer into the back part of the eye (known as “metastasis”)
- Immune-mediated disease—may cause inflammation of blood vessels (known as “vasculitis”) or inflammation of the choroid and/or retina, resulting in separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)
- Unknown cause (idiopathic disease)—common
- Toxicity—antifreeze (ethylene glycol); individual animal reaction to medications (such as trimethoprim-sulfa)
- Trauma

RISK FACTORS

- Feline leukemia virus (FeLV) or feline immunodeficiency virus (FIV) infection may increase the likelihood that a cat will become infected with other disease-causing agents that involve the eye (such as *Toxoplasma*) that cause inflammation of the back part of the eye (choroid and retina)
- Dogs or cats on medications to decrease the immune response for other medical problems



TREATMENT

HEALTH CARE

- Depends on physical condition of patient
- Usually outpatient
- Fluid or other therapy for generalized (systemic) disease



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Identify and treat any underlying, generalized (systemic) disease, such as itraconazole for a fungal infection (known as “systemic mycosis”); doxycycline for rickettsial infection; azithromycin for bartonellosis
- Medications applied directly to the surface of the eye (known as “topical medications”)—are not effective for treatment of chorioretinitis in dogs with intact lenses (the lens [singular] is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye [retina])
- Generalized (systemic) therapy is administered by injection or by mouth (orally)—required for treatment of inflammation of the back part of the eye (choroid and retina; “chorioretinitis”)

- Feline toxoplasmosis—clindamycin for 14 to 21 days
- Systemic steroids administered by mouth (such as prednisone) at anti-inflammatory doses—when generalized (systemic) fungal infection (mycosis) has been ruled out or is being treated with appropriate systemic antifungal therapy; avoid use, unless large areas of the retina are affected and vision is threatened severely
- Systemic steroids administered by mouth (such as prednisone) at doses to decrease the immune response (immunosuppressive doses) for immune-mediated disease; may facilitate separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)
- Topical steroids applied directly to the eye (such as 1% prednisolone acetate or 0.1% dexamethasone) and atropine (1%) to dilate the pupil and reduce pain—for inflammation of the uvea (known as “panuveitis” in which inflammation involves both the front part of the eye, including the iris [known as “anterior uveitis”] and the back part of the eye [choroid])
- Treatment for increased pressure in the eye (glaucoma)—as appropriate for secondary glaucoma in which the pressure within the eye [intraocular pressure] is increased secondary to inflammation in the eye
- Cancer—chemotherapeutic agents
- Uveodermatologic syndrome (a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris [anterior uveitis], inflammation of the posterior uvea or choroid [posterior uveitis], or both and coexistent inflammation of the skin [dermatitis], characterized by loss of pigment in the skin of the nose and lips)—may require azathioprine (a medication that decreases the immune response) and steroids to control inflammation



FOLLOW-UP CARE

PATIENT MONITORING

- As appropriate for underlying cause and type of medical treatment
- Blood work, including a complete blood count (CBC), platelet count and serum biochemistry tests for liver enzymes—if giving azathioprine
- Monitor intraocular pressure (IOP)—for cases with inflammation of the front part of the eye, including the iris (anterior uveitis) to determine if the pressure is increasing and possible glaucoma is developing

PREVENTIONS AND AVOIDANCE

- Tick and flea control measures to prevent infection with various disease-causing agents (such as *Borrelia* that causes Lyme disease)

POSSIBLE COMPLICATIONS

- Permanent blindness
- Cataracts (opacity in the normally clear lens, preventing passage of light to the back part of the eye [retina])
- Glaucoma (increased pressure in the eye)
- Long-term (chronic) eye pain
- Death—secondary to underlying, generalized (systemic) disease

EXPECTED COURSE AND PROGNOSIS

- Prognosis for vision—guarded to good, depending on amount of retina affected; visual deficits or blindness may develop if large areas of the retina were destroyed; localized (focal) disease and multiple areas of disease (multifocal disease) of the retina do not impair vision markedly, but do leave scars
- Prognosis for life—guarded to good, depending on underlying cause



KEY POINTS

- Chorioretinitis may be a sign of a generalized (systemic) disease; therefore, appropriate diagnostic testing is important
- Immune-mediated disease requires lifelong therapy to control inflammation of the back part of the eye (choroid and retina)
- Dogs with uveodermatologic syndrome also may have inflammation of the front part of the eye, including the iris (anterior uveitis) and secondary glaucoma (in which the pressure within the eye [intraocular pressure] is increased secondary to inflammation in the eye), which require treatment; inflammation of the skin (dermatitis) also may require management

CHYLOTHORAX



BASICS

OVERVIEW

- “Chylo-” refers to chyle; “thorax” refers to the chest
- “Chyle” is a milky to slightly yellow fluid composed of lymph and fats taken up from the intestines and eventually transferred to the circulation through the thoracic duct; “lymph” is a watery fluid that contains white-blood cells that travels through lymphatic vessels—it transports lymphocytes (a type of white-blood cell) and fats from the small intestines to the blood stream; the “thoracic duct” is the main lymph vessel of the body—it crosses the chest near the spine, and empties into the venous circulation
- “Chylothorax” is an accumulation of chyle in the space between the chest wall and lungs (known as the “pleural space”)
- “Lymphangiectasia” is defined as the dilation of the lymphatic vessels; it results from blockage or obstruction of the lymphatic vessels
- Lymphangiectasia in the chest (known as “thoracic lymphangiectasia”)—tortuous, dilated lymphatic vessels found in many animals with accumulation of chyle in the space between the chest wall and lungs (chylothorax)
- Inflammation of the tissue lining the chest cavity and covering the lungs characterized by the development of scar tissue (known as “fibrosing pleuritis”)—condition in which thickening of the tissue lining the chest cavity and covering the lungs (known as the “pleura”) leads to constriction of the lung lobes; when severe, it results in marked restriction of breathing; may be caused by any long-term (chronic) build-up of inflammatory fluid in the space between the chest wall and lungs (known as “pleural exudate”), but is most commonly associated with accumulation of chyle (chylothorax) or accumulation of pus (known as “pyothorax”)

GENETICS

- Unknown

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dogs—Afghan hounds and shiba inus
- Cats—Asian breeds (such as the Siamese and Himalayan) appear to have a higher number of cases than other breeds

Mean Age And Range

- Any age may be affected
- Afghan hounds—develop when middle-aged
- Shiba inus—develop when young (less than 1 to 2 years of age)
- Cats—old animals may be more likely to develop condition than young cats; may indicate an association with cancer

Predominant Sex

- None identified

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary, depending on the underlying cause, rapidity of fluid accumulation, and volume of fluid
- Usually not exhibited until marked impairment of breathing
- Many patients appear to have condition for prolonged periods before diagnosis; they probably reabsorb the milky fluid (chyle) at a rate that prevents obvious breathing impairment
- Difficulty breathing (known as “dyspnea”) or coughing; coughing may have been present for months before examination
- Rapid breathing (known as “tachypnea”)
- Depression
- Lack of appetite (known as “anorexia”) and weight loss
- Exercise intolerance
- Muffled heart and lung sounds detected when listening to the chest with a stethoscope (known as “auscultation”)
- Increased lung sounds, particularly in the lung fields near the animal’s back
- Pale gums and moist tissues of the body (known as “mucous membranes”)
- Bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”)
- Irregular heartbeats (known as “arrhythmias”)
- Heart murmur
- Detectable pulses in the jugular veins in association with right-sided heart failure
- Decrease in the ability to gently compress the front part of the chest—common in cats with a mass in the front of the mediastinum and fluid build-up in the space between the chest wall and lungs (known as “pleural effusion”); the “mediastinum” is the center portion of the chest that contains the heart and other organs (except for the lungs)

CAUSES

- Unknown cause (so called “idiopathic chylothorax”)—most patients
- Masses in the front of the mediastinum (the center portion of the chest that contains the heart and other organs [except for the

lungs])—mediastinal lymphoma (lymphoma is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body); thymoma (tumor that arises from the thymus)

- Heart disease—disease of the heart muscle (known as “cardiomyopathy”); fluid build-up between the heart and the sac surrounding the heart (known as “pericardial effusion”); heartworm infection; heart birth defects
- Nodular lesions caused by infection with a fungus (known as “fungal granulomas”)
- Blood clots in veins
- Congenital (present at birth) abnormality of the thoracic duct (the main lymph vessel of the body)
- Heart surgery

RISK FACTORS

- Unknown



TREATMENT

HEALTH CARE

- Patients with difficulty breathing (dyspnea) with suspected fluid build-up between the chest wall and lungs (pleural effusion)—immediate medical procedure to tap the chest (known as “thoracocentesis”); removal of even small amounts of pleural effusion may improve breathing markedly
- Identify and treat the underlying cause, if possible
- Medical management—usually outpatient with intermittent procedures to tap the chest (thoracocentesis), as necessary to prevent difficult breathing (dyspnea)
- Chest tubes—placed in patients with suspected chylothorax secondary to trauma (very rare), with rapid fluid accumulation, or after surgery
- Unsuccessful medical management (try 2 to 3 months)—consider surgery
- Patients may become debilitated if procedures to tap the chest (thoracocentesis) are performed frequently; attention to diet is important
- Chest taps (thoracocentesis)—perform under sterile conditions to reduce the risk of introducing infection into the chest; antibiotics generally are unnecessary if sterile technique is used

ACTIVITY

- Patients usually will restrict their own exercise as the fluid volume in the space between the chest wall and lungs increases or if they develop fibrosing pleuritis (inflammation of the tissue lining the chest cavity and covering the lungs characterized by the development of scar tissue)

DIET

- Low-fat diet—may decrease the amount of fat in the fluid build-up in the space between the chest wall and lungs (pleural effusion), which may improve the patient’s ability to resorb fluid from the chest cavity; not a cure; may help in management
- Medium-chain triglycerides—once thought to be absorbed directly into the system of veins that carry blood from the abdominal organs to the liver (known as the “portal system”), bypassing the thoracic duct (the main lymph vessel of the body); recent information shows that medium-chain triglycerides actually are transported via the thoracic duct of dogs; therefore, they are less useful than previously believed

SURGERY

Thoracic Duct Ligation and Surgical Removal of Part of the Sac Around the Heart (known as “Pericardectomy”)

- Recommended in patients that do not respond to medical management
- The thoracic duct (the main lymph vessel of the body) usually has multiple branches in the back part of the chest, where the surgical procedure to “tie off” or “ligate” the thoracic duct is performed; failure to ligate all branches results in continued fluid build-up in the space between the chest wall and lungs (pleural effusion)
- Injection of methylene blue dye greatly facilitates visualization and complete ligation of all branches of the thoracic duct
- Thickening of the sac around the heart (known as the “pericardium”)—perform surgical removal of part of the sac around the heart (known as a “pericardectomy”) simultaneously with tying off or ligation of the thoracic duct

Other Surgical Considerations

- Thoracic duct ligation not successful—may consider procedures in which the flow of lymph is shunted into another part of the body
- Extensive fibrosing pleuritis (inflammation of the tissue lining the chest cavity and covering the lungs characterized by the development of scar tissue)—makes surgery harder, but does not appear to affect prognosis, if fluid build-up can be stopped



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Rutin, a bioflavonoid—complete resolution of fluid build-up (effusion) appears to occur in some patients; further study is required to determine whether resolution occurs spontaneously or in response to this therapy
- Somatostatin (octreotide [Sandostatin®]), an inhibitory hormone—a naturally occurring substance that inhibits secretions of the stomach, jejunum (middle section of the small intestines), pancreas and inhibits secretion of bile by the liver; prolongs movement of food and fluids through the stomach and intestines (known as “gastrointestinal transit time”), and stimulates water absorption in the intestines; resolution of fluid build-up (pleural effusion) has occurred in dogs and cats with chylothorax of unknown cause (idiopathic chylothorax) treated with octreotide



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor closely for difficulty breathing (dyspnea); perform procedures to tap the chest (thoracocentesis) as needed
- Resolution (spontaneously or following surgery)—periodically re-evaluate for several years to detect recurrence

POSSIBLE COMPLICATIONS

- Fibrosing pleuritis (inflammation of the tissue lining the chest cavity and covering the lungs characterized by the development of scar tissue)—most common serious complication of long-term (chronic) disease
- Decreased ability to develop a normal immune response (known as “immunosuppression”)—caused by decreased number of lymphocytes; “lymphocytes” are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process—may develop in patients undergoing repeated and frequent procedures to tap the chest (thoracocentesis)
- Low levels of sodium in the blood (known as “hyponatremia”) and high levels of potassium in the blood (known as “hyperkalemia”)—documented in affected dogs undergoing multiple procedures to tap the chest (thoracocentesis)

EXPECTED COURSE AND PROGNOSIS

- May resolve spontaneously or after surgery
- Untreated or long-term (chronic) disease—may result in severe fibrosing pleuritis (inflammation of the tissue lining the chest cavity and covering the lungs characterized by the development of scar tissue) and persistent difficulty breathing (dyspnea)
- Euthanasia—frequently performed in patients that do not respond to medical management or surgery



KEY POINTS

- No treatment will stop the fluid build-up (effusion) in all patients with chylothorax of unknown cause (idiopathic chylothorax)
- The condition may resolve spontaneously in some patients after several weeks or months

FIBROSIS AND CIRRHOSIS OF THE LIVER



BASICS

OVERVIEW

- The liver is the largest gland in the body; it has many functions, including production of bile (a fluid substance involved in digestion of fats); bile ducts begin within the liver itself as tiny channels to transport bile—the ducts join together to form larger bile ducts and finally enter the extrahepatic or common bile duct, which empties into the upper small intestine; the system of bile ducts is known as the “biliary tree”
- The gallbladder is the storage unit for bile; bile is stored until it is needed for fat digestion
- “Fibrosis of the liver” involves the formation of scar tissue that replaces normal liver tissue
- “Cirrhosis of the liver” is generalized (diffuse) formation of scar tissue (fibrosis), associated with regenerative nodules and deranged liver architecture

GENETICS

- Familial (runs in certain families or lines of animals) susceptibility for long-term (chronic) inflammation of the liver (condition known as “chronic hepatitis”)—Doberman pinschers, cocker spaniels, Labrador retrievers

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Any breed or mixed-breed dog
- Copper-storage liver disease (known as “copper-storage hepatopathy”)—Bedlington terriers, Dalmatians, possibly some Doberman Pinschers, Labrador retrievers
- Scarring of the liver in young dogs of unknown cause (so called “juvenile idiopathic hepatic fibrosis”)—German shepherd dogs, standard poodles
- Uncertain disorders—West Highland white terriers, Skye terriers
- Cats—long-term [chronic] inflammation of the bile duct or biliary tree (known as “cholangitis”) and inflammation of the bile ducts and liver (known as “cholangiohepatitis”) leading to blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction) may develop progressive damage and scarring of the biliary tree and liver (known as “biliary cirrhosis”)

Mean Age and Range

- Cirrhosis (dogs)—any age; common in middle to old age
- Copper-storage liver disease (copper-storage hepatopathy) and scarring of the liver of unknown cause (idiopathic hepatic fibrosis)—young/middle-aged adults
- Progressive damage and scarring of the biliary tree and liver (biliary cirrhosis) in cats with long-term (chronic) inflammation of the bile ducts and liver (cholangiohepatitis)—greater than 7 years of age

Predominant Sex

- Cocker spaniels—males are two-to-eight times more likely to develop fibrosis and cirrhosis of the liver than females
- Doberman pinschers and Labrador retrievers—may be more common in females than in males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Initially—vague and nonspecific signs
- Later—relate to complications of increased blood pressure within the portal vein (the vein carrying blood from the digestive organs to the liver; condition known as “portal hypertension”) and to impaired liver function; signs include nervous system signs (such as seizures, blindness) caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”); fluid build-up in the abdomen (known as “ascites”); bleeding in the stomach and/or upper small intestine
- Long-term (chronic) intermittent sluggishness (lethargy), lack of appetite (known as “anorexia”), reduced or poor body condition
- Gastrointestinal signs: vomiting, diarrhea or constipation
- Black, tarry stools due to the presence of digested blood (known as “melena”); late stage of disease
- Increased thirst (known as “polydipsia”) and increased urination (known as “polyuria”)
- Late onset—fluid build-up in the abdomen (ascites); yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”); bleeding; nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (hepatic encephalopathy)
- Cats—fluid build-up in the abdomen (ascites) uncommon; drooling (known as “ptyalism”) with nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (hepatic encephalopathy)
- Liver size—small liver size in dogs; variable liver size in cats
- Bleeding tendencies (uncommon)
- Skin lesions with superficial, ulcerative inflammation (known as “superficial necrolytic dermatitis”)—not true cirrhosis

CAUSES

- Long-term (chronic) liver injury

- Long-term (chronic) inflammatory bowel disease (IBD)
- Drug- or toxin-induced liver injury—copper-storage liver disease (copper-storage hepatopathy); medications to control seizures (known as “anticonvulsants”); azole medications to treat fungal infections; medication to treat intestinal parasites (oxibendazole); antibiotic (trimethoprim-sulfamethoxazole); nonsteroidal anti-inflammatory drugs (NSAIDs); long-term (chronic) food borne toxin (aflatoxins)
- Infections disease—leptospirosis, canine adenovirus-1 infection
- Long-term (chronic) inflammation of the bile ducts and liver (known as “cholangiohepatitis”) in cats
- Long-term (chronic) blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction)—lasting more than 6 weeks—in dogs or cats
- Single episode of massive tissue death in the liver (known as “hepatic necrosis”)

RISK FACTORS

- Breed predisposition: copper toxicity to liver cells (known as “hepatotoxicity”) or other causes yet ill-defined
- Long-term (chronic) inflammation of the liver and bile ducts
- Accumulation of copper or iron in the liver
- Blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction)
- Long-term (chronic) administration of phenobarbital (medication to control seizures) in dogs



TREATMENT

HEALTH CARE

- Outpatient—patients with minimal signs that are eating
- Inpatient—diagnostic tests; treatment for dehydration, lack of appetite (anorexia), severe hepatic encephalopathy, or bleeding in the intestines due to blood vessel disease caused by high blood pressure (known as “hypertensive vasculopathy”)
- Fluids
- B-complex vitamins (especially for cats)
- Glucose—if patient has low blood sugar (known as “hypoglycemia”)
- Potassium chloride—in fluids, as needed
- Tap the abdomen and remove excess fluid (procedure known as “abdominocentesis”) as treatment for tense abdomen, causing signs (such as difficulty breathing); used in cases that do not respond to medical treatments to remove the excess fluid

ACTIVITY

- Limit activity

DIET

- Withhold food in cases of sudden (acute), severe hepatic encephalopathy (such as those with stupor or coma) or vomiting associated with intestinal bleeding or inflammation of the pancreas (pancreatitis)
- Hepatic encephalopathy (nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia): restrict protein intake, use soy or dairy protein sources (dogs) combined with medical treatment to increase nitrogen tolerance; individualize protein intake (maintain body condition, maintain albumin [type of protein] levels, control hepatic encephalopathy)
- Sodium restriction if patient has fluid build-up in the abdomen (ascites)
- Fat restriction rarely needed
- Supplement water-soluble vitamins (such as vitamin B)

SURGERY

- Cirrhosis—high anesthetic risks; gas anesthetics preferred—isoflurane or sevoflurane
- Blood-clotting disorder (known as “coagulopathy”)—increased likelihood of bleeding, with even minor surgeries
- Postoperative intensive care—critical to avoid hepatic encephalopathy (nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia), maintain hydration and normal blood sugar
- Susceptible to bacterial infection—administer appropriate antibiotics



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treatments for specific etiologies: chelate copper if patient has copper-storage liver disease: to “chelate copper” is to use specific chemicals to tie up copper in the system and to allow it to be removed from the body
- Discontinue medications that potentially may be toxic to the liver, as directed by your pet’s veterinarian

- Long-term inflammation of the liver (chronic hepatitis)—medications to alter the immune response (known as “immune modulation”): steroids (prednisolone, prednisone); azathioprine (a chemotherapeutic drug used to decrease the immune response) in dogs (may be used in combination with prednisone, antioxidants [such as S-adenosylmethionine or SAMe, vitamin E], and medication to decrease scarring of the liver [known as “antifibrotics,” such as ursodiol, polyunsaturated phosphatidylcholine, colchicine])
- Medications to protect the liver—ursodeoxycholate, Vitamin E, SAMe; elemental zinc
- Medications to protect the lining of the stomach
- Eliminate intestinal parasites
- Medications to remove excess fluid from the body (known as “diuretics”) may be used in patients with fluid build-up in the abdomen (ascites)—furosemide, spironolactone Dexamethasone is a steroid that may be used in patients with fluid build-up in the abdomen (ascites) instead of prednisone
- Mycophenolate is a potential alternative for azathioprine



FOLLOW-UP CARE

PATIENT MONITORING

- Blood work (including liver enzymes, albumin, blood urea nitrogen, cholesterol)—monthly or quarterly, depends on patient status
- Serial monitoring of total serum bile acids (bile acid levels are used to monitor liver function)
- Body condition score (estimate of weight status [under or overweight] as compared to normal weight) and muscle mass—reflects nutritional adequacy/nitrogen balance
- Monitor size of the abdomen: reflects volume of fluid build-up in the abdomen (ascites)
- Patients treated with azathioprine, mycophenolate or colchicine—monitor for possible bone-marrow toxicity using frequent complete blood counts (CBCs)

PREVENTIONS AND AVOIDANCE

- Reduce factors that may increase the likelihood of hepatic encephalopathy (nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)—dehydration; infection; breakdown of lean muscle mass and body tissues (known as “catabolism”); low blood potassium (known as “hypokalemia”); high protein meals; intestinal parasites; bleeding in the intestines; certain drugs

POSSIBLE COMPLICATIONS

- Hepatic encephalopathy (nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia), generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”), bleeding—may be life threatening
- Blood-clotting disorder (known as disseminated intravascular coagulopathy” or “DIC”)—may be a terminal event

EXPECTED COURSE AND PROGNOSIS

- Occasional flare-ups of hepatic encephalopathy (nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia) and fluid build-up in the abdomen (ascites) may require hospitalization for adjustment of diet and medical treatment; sodium restriction and medications to remove excess fluid from the body (diuretics) may require titration to achieve optimal control of fluid build-up in the abdomen (ascites)
- Course of fibrotic/cirrhotic liver disease is poorly characterized; fibrosis severity and bridging fibrosis associated with significantly shorter survival (one study: dogs)
- Scarring of the liver in young dogs of unknown cause (so called “juvenile idiopathic hepatic fibrosis”) in dogs—survival up to 6 years
- Cirrhosis—survival greater than 5 years with aggressive medical treatment



KEY POINTS

- “Fibrosis of the liver” involves the formation of scar tissue that replaces normal liver tissue
- “Cirrhosis of the liver” is generalized (diffuse) formation of scar tissue (fibrosis), associated with regenerative nodules and deranged liver architecture
- Treatment is designed to control signs and improve the patient’s condition, but not to cure (known as “palliative treatment”)
- Reduce factors that may increase the likelihood of hepatic encephalopathy (nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)—dehydration; infection; breakdown of lean muscle mass and body tissues (catabolism); low blood potassium (hypokalemia); high protein meals; intestinal parasites; bleeding in the intestines; certain drugs

CLOSTRIDIAL ENTEROTOXICOSIS

(DIARRHEA RELATED TO AN INTESTINAL TOXIN PRODUCED BY A BACTERIA, *CLOSTRIDIUM PERFRINGENS*)



BASICS

OVERVIEW

- A complex syndrome characterized by diarrhea in dogs and cats associated with a particular bacteria, *Clostridium perfringens* (abbreviated “CP”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Suspected that up to 15% to 20% of diarrhea cases in dogs is CP-related; less common in cats

Mean Age and Range

- Disease may occur in any age animal; most animals that develop chronic clinical signs tend to be middle-aged or older

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical syndromes are associated with sudden (acute) self-limiting diarrhea, lasting for 5 to 7 days; chronic intermittent diarrhea; or signs associated with other gastrointestinal or non-gastrointestinal disease
- Chronic signs often are characterized by intermittent episodes recurring every 2 to 4 weeks that may persist for months to years; the syndrome may result as a hospital-acquired disease (known as “nosocomial disease”) with signs precipitated during or shortly following hospitalization or boarding at a kennel
- CP has been associated with sudden bloody stomach and intestinal inflammation (acute hemorrhagic gastroenteritis) and observed with cases having parvovirus
- Most common sign is large-bowel diarrhea characterized by fecal mucus, small amounts of fresh blood, small scant stools, straining to defecate (known as “tenesmus”) with increased frequency of stools
- Dogs may have signs of small-bowel diarrhea characterized by a large volume of watery stool
- Other signs include vomiting, passing gas (flatulence), abdominal discomfort, or a generalized unthriftiness (lack of thriving) in chronic cases
- Evidence of systemic illness or debilitation is rare; abdominal discomfort may be detected when the veterinarian feels the abdomen (palpation) during the physical examination; fever is uncommon

CAUSES

- It is unknown if the intestinal toxin-producing (enterotoxigenic) CP is a true infection or if the bacteria normally is present in the animal’s system and due to certain conditions (such as stress), it produces the intestinal toxin and causes disease (known as an “opportunistic pathogen”)
- Only certain strains of CP are capable genetically of producing the intestinal toxin and only certain animals are affected clinically; the disease may be associated with the development of an excessive number of bacteria in the small intestine (a condition known as “small intestinal bacterial overgrowth”)
- Diarrhea may be associated with dietary indiscretions or diet change

RISK FACTORS

- Stress factors to the gastrointestinal tract, dietary change, other disease conditions existing at the same time, or hospitalization may precipitate signs
- The ability of CP to cause disease (known as the “pathogenicity”) may depend on the status of the gastrointestinal tract, including its metabolic, mucosal, and immunologic integrity
- Possibly related to a decrease in Immunoglobulin A (known as “IgA deficiency”) in which a normal immunity-related protein in the intestinal tract is too low to be effective in protecting the intestinal tract from infection and/or the toxin
- A higher pH (known as being alkaline) within the intestinal tract promotes bacteria to produce spores (sporulation) and toxins (enterotoxin production)
- Primary intestinal bacterial overgrowth



TREATMENT

HEALTH CARE

- Most treated as outpatients
- Hospitalization may be required when diarrhea or vomiting is severe, leading to dehydration and electrolyte imbalance

- Fluid and electrolyte therapy may be required to replace losses occurring from diarrhea

ACTIVITY

- Restricted during sudden (acute) disease

DIET

- Diet plays a role in the treatment and management of cases with chronic recurring disease; high fiber diets (either soluble/fermentable or insoluble fiber) often result in clinical improvement by reducing the number of clostridial bacteria in the intestinal tract and by acidifying the distal intestine, thus limiting the production of spores and toxins of CP
- Commercial high-fiber diets can be supplemented with psyllium as a source of soluble fiber under the direction of your pet's veterinarian
- Diets low in fiber should be supplemented with fiber (such as coarse bran) as a source of insoluble fiber or psyllium as a source of soluble fiber under the direction of your pet's veterinarian
- Prebiotic diets containing fermentable substances (such as fructo-oligosaccharides) may be beneficial by changing the make up of the bacteria in the intestinal tract



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Sudden (acute) self-limiting disease usually requires antibiotics for 5 to 7 days; most patients respond well to antibiotic therapy (such as oral ampicillin or amoxicillin, clindamycin, metronidazole, or tylosin)
- Chronic recurring cases often require prolonged antibiotic therapy; tylosin mixed with the food or formulated in capsules is suggested for long-term management
- High doses of antibiotics may not be necessary to prevent recurrence in chronic cases; low antibiotic levels may not actually reduce the number of CP bacteria in the intestine, but may change the microenvironment, preventing the bacteria from producing spores (sporulation) and toxins (enterotoxin production); antibiotic resistance may develop
- Probiotics (such as lactobacillus) may have antibacterial effects on *Clostridium* and some reports suggest a benefit may be seen in chronic cases
- Chronic cases may respond well to high-fiber diets and changing the diet may be attempted as the sole therapy following resolution of signs



FOLLOW-UP CARE

PATIENT MONITORING

- The patient's response to therapy supports the diagnosis, and rarely are repeated diagnostics necessary

PREVENTIONS AND AVOIDANCE

- Infection is associated with environmental contamination; disinfecting the environment is difficult
- Feeding high-fiber diets may decrease the incidence of hospital- or boarding kennel-acquired diarrhea

EXPECTED COURSE AND PROGNOSIS

- Most animals respond well to therapy
- Chronic cases may require long-term therapy to control clinical signs
- Failure in response suggests other existing disease conditions and further diagnostic evaluation is needed



KEY POINTS

- A complex syndrome characterized by diarrhea in dogs and cats associated with a particular bacteria, *Clostridium perfringens* (abbreviated "CP")
- Suspected that up to 15-to-20% of diarrhea cases in dogs is CP-related; less common in cats
- Clinical syndromes are associated with sudden (acute) self-limiting diarrhea, lasting for 5 to 7 days; chronic intermittent diarrhea; or signs associated with other gastrointestinal or non-gastrointestinal disease
- Sudden (acute disease) is often self-limiting, while chronic cases may require prolonged therapy

DISEASE CAUSED BY *COCCIDIOIDES*, A TYPE OF FUNGUS (COCCIDIOIDOMYCOSIS)



BASICS

OVERVIEW

- Coccidioidomycosis is a generalized (systemic) disease caused by inhalation of infective spores of the soil-borne fungus, *Coccidioides immitis*
- “Mycosis” is the medical term for any disease caused by a fungus

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Most patients are young animals (less than 4 years of age)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Lack of appetite (known as “anorexia”)
- Coughing
- Fever, unresponsive to antibiotics
- Lameness
- Weakness, partial paralysis, back and neck pain
- Seizures
- Change in vision
- Weight loss

Dogs

- Coughing
- Difficulty breathing (known as “dyspnea”)
- Fever
- Bone swelling, joint enlargement, and lameness
- Extreme weight loss with muscle wasting (known as “cachexia”)
- Sluggishness (lethargy)
- Enlarged lymph nodes (known as “lymphadenomegaly”)
- Skin ulcers and draining tracts
- Inflammation of the iris (the colored part of the eye) and other areas in the front part of the eye (known as “uveitis”); inflammation of the cornea (known as “keratitis”); inflammation of the iris (known as “iritis”)

Cats

- Extreme weight loss with muscle wasting (cachexia)
- Draining skin lesions
- Difficulty breathing (dyspnea)
- Lameness caused by bone involvement
- Inflammation of the iris (the colored part of the eye) and other areas in the front part of the eye (uveitis)

CAUSES

- Infection by the soil-borne fungus, *Coccidioides immitis*
- *Coccidioides immitis* grows several inches deep in the soil, where it survives high environmental temperatures and low moisture; after a period of rainfall, the fungus returns to the soil surface where it forms spores, which are released and spread by wind and dust storms

RISK FACTORS

- *Coccidioides immitis* is found in the southwestern United States in the geographic Lower Sonoran life zone—it is more common in Southern California, Arizona, and southwest Texas, and less common in New Mexico, Nevada, and Utah
- Aggressive nosing about in soil and underbrush may expose susceptible animals to large doses of the fungus in contaminated soil
- Dust storms after the rainy season; increased number of cases are noted after earthquakes
- Land development (where much earth disruption occurs) may lead to increased exposure



TREATMENT

HEALTH CARE

- Generally treated as outpatients
- Clinical signs (such as seizures, pain, coughing) should be treated appropriately

ACTIVITY

- Restrict activity until clinical signs begin to subside

DIET

- Feed a high-quality palatable diet to maintain body weight

SURGERY

- Surgical removal of an affected organ may be indicated for cases with localized, nodular involvement in various organs (such as a consolidated lung lobe or involvement of the eye or kidney)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treatment of widespread (disseminated) disease often requires at least one year of aggressive antifungal therapy
- Low-dose, short-term treatment with steroids (prednisone administered by mouth) and cough suppressants may be required to alleviate the respiratory signs

Dogs

- Several antifungal medications that may be given by mouth (oral treatment) currently are available for the treatment of coccidioidomycoses; they include 1) fluconazole—noted to greatly increase the success of treatment; has been used in treating infections involving the nervous system; cost of the drug has decreased significantly with the availability of a medical grade generic compound; 2) ketoconazole—may be given with food; some believe that administration of high doses of vitamin C at the time that ketoconazole is administered may improve the absorption of the drug; treatment should be continued for 1 year; 3) itraconazole—administered similarly as ketoconazole; it has been reported to have a higher penetration rate than ketoconazole, but a better clinical response has not been observed
- Amphotericin B is an antifungal medication that must be administered by intravenous injection; it is recommended rarely because of the high risk of kidney damage and the availability of effective oral medications

Cats

- Any of the following antifungal medications may be used in cats: ketoconazole, itraconazole, fluconazole



FOLLOW-UP CARE

PATIENT MONITORING

- Serologic tests (blood tests that detect the presence of antibodies to a certain disease-causing agent or antigen; an “antibody” is a protein that is produced by the immune system in response to a specific antigen)—should be monitored every 3 to 4 months; animals should be treated until their antibody titers fall to less than 1:4
- Animals displaying poor response to therapy should have a 2 to 4 hour post-pill drug level measured to ensure adequate absorption of the drug
- Blood urea nitrogen (“BUN,” a blood test used in assessing the kidneys) and urinalysis should be monitored in all animals treated with amphotericin B; treatment should be discontinued temporarily if the BUN rises above 50 mg/dl or if granular casts are noted in the urine

PREVENTIONS AND AVOIDANCE

- No vaccine is available for dogs or cats
- Contaminated soil in areas where *Coccidioides immitis* are found (known as “endemic areas”) should be avoided, particularly during dust storms after the rainy season

POSSIBLE COMPLICATIONS

- Lung disease resulting in severe coughing may worsen temporarily after treatment is started, owing to inflammation in the lungs
- Liver toxicity may result from ketoconazole treatment
- Kidney toxicity may result from amphotericin B treatment

EXPECTED COURSE AND PROGNOSIS

- Coccidioidomycosis is considered one of the most severe and life threatening of the generalized (systemic) fungal diseases (mycoses)
- The prognosis is guarded to grave
- Many dogs will improve following oral antifungal medication therapy; however, relapses may be seen, especially if therapy is shortened
- The overall recovery rate has been estimated at 60%, but some report a 90% response to fluconazole therapy
- The prognosis for cats is not well documented, but rapid spread of the disease throughout the body (dissemination) requiring long-term therapy should be anticipated
- Serologic tests (blood tests that detect the presence of antibodies to a certain disease-causing agent or antigen; an “antibody” is a protein that is produced by the immune system in response to a specific antigen) every 3 to 4 months after completion of treatment is recommended to monitor the possibility of relapse
- Spontaneous recovery from widespread (disseminated) coccidioidomycosis without treatment is extremely rare



KEY POINTS

- The necessity and expense of long-term treatment of this serious illness, with the possibility of treatment failure, should be discussed with your pet's veterinarian
- The antifungal medications used in treatment have potential side effects

COGNITIVE DYSFUNCTION SYNDROME



BASICS

OVERVIEW

- Syndrome associated with brain aging
- Leads to changes in the pet's awareness, decreased responsiveness to stimuli, and deficits in learning and memory
- Pet may have increasing signs of anxiety with advancing age
- Subtle signs are seen in early stages, referred to as "cognitive decline"

GENETICS

- Genetic correlation with respect to the distribution of beta-amyloid in the brain and the age at which it begins to accumulate

SIGNALMENT/DESCRIPTION of ANIMAL

- Dogs and cats
- More common with increasing age
- A decline in memory and learning can be seen in dogs as early as 6 years of age
- Clinical signs in cats may develop at a slightly older age
- Deficits may not be noticed by pet owners until several years later, except in dogs trained to perform more specialized tasks (such as hearing ear, seeing eye, drug detection, agility)

SIGNS/OBSERVED CHANGES in the ANIMAL

Historical Findings

Most clinical signs can be placed into 5 categories:

- Disorientation, including getting lost in familiar environments, confusion, or inability to navigate through familiar routes (such as going to the wrong side of door)
- Interactions with humans or other animals may be altered (possible decline in play, increased/decreased interest in affection, or an increase in irritability)
- Sleep-wake cycle alterations (temporal disorientation), including night waking or vocalization and perhaps an increase in sleep during the day
- Housetraining and other previously learned behaviors might deteriorate; house soiling, lack of response to previously learned commands, or becoming less adept at performing learned tasks (such as agility, working ability) may occur
- Activity may be altered—inactivity, less interest in exploration, self-care, or even eating; as the condition progresses, activity levels may increase with signs of restlessness, pacing, aimless wandering, or compulsive activity disorders (such as excessive licking)
- Anxiety and agitation may increase in pets with cognitive dysfunction

Physical Examination

- No specific abnormalities related to Cognitive Dysfunction Syndrome are seen; pet may have non-related physical changes or health concerns

CAUSES

- Exact cause is unknown and animals are variably affected
- Genetic factors may predispose pets to developing cognitive decline

RISK FACTORS

- Chronic or recurrent illness or stress might lead to increased accumulation of toxic free radicals in the brain
- Conditions that affect the blood supply to the brain (such as systemic high blood pressure [hypertension], low red blood cell count [anemia])



TREATMENT

HEALTH CARE

- Outpatient care
- Depends on the type and severity of the clinical signs of cognitive dysfunction

ACTIVITY

- Maintain as much exercise, play, training, work, and other daily routines as is practical for the pet's age and health
- Providing mental and physical stimulation has been shown to reduce the chance of cognitive decline

DIET

- Selected based on the pet's overall health assessment
- If the pet's overall health does not require a special therapeutic diet, then an antioxidant-fortified senior diet (e.g., Hill's Prescription Diet® b/d®) should be utilized
- Hill's Prescription Diet b/d has been shown to improve memory, learning ability, and clinical signs of Cognitive Dysfunction Syndrome
- Natural supplements with combinations of antioxidants, phosphatidylserine and DHA may be useful if diet cannot be changed; talk to your veterinarian before adding supplements to your pet's diet



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Selegiline

- Licensed for use in dogs in North America
- Monoamine oxidase (MAO) B inhibitor, in dogs, may contribute to improved transmission of brain chemicals, lead to a decrease in free radicals, and have a protective effect for nerve cells in the brain
- Reevaluate clinical signs for improvement after 1 to 2 months
- Side effects might include occasional gastrointestinal upset and restlessness, and repetitive behavior at higher doses

Nicergoline

- Not licensed for use in dogs in North America, but is licensed in other countries
- Used in elderly dogs with decreased activity, sleep disorders, decreased exercise tolerance, house soiling (including incontinence), reduced appetite, and decreased awareness
- May increase blood flow in the brain, may contribute to improved transmission of brain chemicals, and have a protective effect for nerve cells in the brain

Propentofylline

- Not licensed for use in dogs in North America, but is licensed in other countries
- Reported to inhibit platelet aggregation and clot (thrombus) formation and increase blood flow
- For use in the treatment of dullness and lethargy in old dogs
- May increase oxygen supply to the central nervous system without increasing glucose demand

General Comment Regarding Cats

- No drugs are approved by the FDA for the treatment of Cognitive Dysfunction Syndrome in cats; your veterinarian will discuss the risks and benefits of medical treatment
- Selegiline has been used and might be effective in cats with anxiety, decreased responsiveness to stimuli, nighttime activity and vocalization, and decreased grooming and appetite

Other Drugs

- Adrafinil or modafinil to improve alertness and exploration
- Anti-inflammatory medication, hormone replacement therapy, and ginkgo extract might be considered based on preliminary work in other species
- Medication used in humans for Alzheimer's disease might be considered in refractory cases; potential side effects include nausea, vomiting, diarrhea, and sleep-wake disturbances
- Anxiety-decreasing drugs (anxiolytics), such as buspirone; drugs to help induce sleep, such as benzodiazepines; or antidepressants, such as fluoxetine (but not in combination with selegiline) might be considered to treat anxiety and apathy
- Homeopathic and natural supplements might help to normalize sleep-wake cycles or reduce anxiety (e.g. DAP pheromone, melatonin, valerian, Bach's flower remedies)



FOLLOW-UP CARE

PATIENT MONITORING

- If a diet or medication is dispensed, then response to therapy should be evaluated after 30 to 60 days and the dose adjusted or treatment changed if the pet has insufficient improvement
- If the pet is stable, twice-yearly checkups are recommended for senior pets unless new problems arise before a reassessment is due

PREVENTIONS AND AVOIDANCE

- Maintaining a stimulating environment and as much activity as is practical for the pet's age and health may help to prevent or delay the onset of cognitive decline
- Early intervention is the best way to slow the progression of cognitive dysfunction

EXPECTED COURSE AND PROGNOSIS

- Diet and medication should control the clinical signs and slow progression in a majority of cases
- Cognitive decline may advance and other health problems are likely to arise despite medical intervention because of the pet's increasing age



KEY POINTS

- Realistic expectations must be understood; treatment is aimed at slowing the progression of the disease, not at curing the pet
- Signs are generally progressive
- Lifelong therapy is required
- Additional medications may be necessary if the pet has multiple health problems
- Any changes in the pet's health or behavior should be reported to your veterinarian immediately, as this may be due to cognitive dysfunction or the emergence of new health problems

DISEASE CAUSED BY *E. COLI*, A TYPE OF BACTERIA (COLIBACILLOSIS)



BASICS

OVERVIEW

- “Colibacillosis” is a disease caused by *Escherichia coli* (*E. coli*)
- *Escherichia coli*—gram-negative bacteria; normal inhabitant of the intestines of most mammals; along with other infectious agents, may increase the severity of parvovirus infections
- Sudden (acute) infection of puppies and kittens in the first week of life; characterized by generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”) and multiple organ involvement
- Infection of old dogs and cats has been documented in the veterinary medical literature
- The female dog is a “bitch;” the female cat is a “queen”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Infection of newborn puppies or kittens (known as “neonatal infection”) is common up to 2 weeks of age
- More common in newborn puppies and kittens less than 1 week of age, which have not received any or adequate amounts of colostrum (the first milk produced at the end of pregnancy that is rich in antibodies; “antibodies” are proteins that are produced by the immune system in response to a specific antigen—when the body is exposed to the antigen, the antibody responds; the “antigen” is any substance that induces an immune response; antigens include proteins, viruses, bacteria, and pollen)
- *E. coli* (usually β -hemolytic)—major cause of generalized disease caused by the spread of bacteria in the blood (septicemia) in newborn puppies exposed while still in the uterus, during birth, or from milk of inflamed mammary glands (condition known as “mastitis”)
- Puppies/kittens and adult animals—sporadic disease often associated with other infectious agents

SIGNS/OBSERVED CHANGES in the ANIMAL

- Newborns—sudden (acute) onset of depression, lack of appetite (known as “anorexia”), vomiting, rapid heart rate (known as “tachycardia”), weakness/lethargy, watery diarrhea, low body temperature (known as “hypothermia”), cold skin, bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); one or more animals affected in a litter
- Puppies/kittens and adults—sudden (acute) vomiting, diarrhea, lack of appetite (anorexia), rapid dehydration, fever

CAUSES

- *E. coli*—one of the most common causes of generalized disease caused by the spread of bacteria in the blood (septicemia) and death in puppies and kittens
- *E. coli* is a normal bacteria found in the adult’s gastrointestinal tract, prepuce, and vagina
- Often found in old dogs and cats at the same time as other infectious agents

RISK FACTORS

Newborns

- Bitch/queen in poor health and nutritional status—unable to provide good care and colostrum (first milk) to offspring
- Lack of colostrum (first milk) or insufficient colostrum
- Dirty birthing environment
- Difficult or prolonged labor and birth
- Crowded facilities—build-up of feces in environment, greater chance for spread of infection

Puppies/Kittens and Adults

- Coexistent disease—parvovirus; heavy parasitism
- Antibiotic treatment—alters normal bacteria of gastrointestinal tract
- Inability to develop a normal immune response (known as “immunosuppression”)
- Inflammation of the mammary glands or breasts of the bitch or queen following birth (known as “post-parturient mastitis”)
- Placement of an intravenous catheter



TREATMENT

HEALTH CARE

- Suddenly (acutely) ill puppies/kittens—inpatients; good nursing care
- Balanced fluids (such as lactated Ringer's solution) administered by injection—restore fluid balance
- Glucose solution specifically designed for treatment of diarrhea, administered by mouth as required

ACTIVITY

- Suddenly (acutely) ill, immature puppies/kittens (that have bacteria in their blood [known as “bacteremia”] or have generalized disease caused by the spread of bacteria in the blood [septicemia])—restricted activity, cage rest, monitoring, and warmth

DIET

- Puppies—likely to be nursing when affected; good nursing care needed with bottle-feeding and/or intravenous nutrients



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotic treatment for generalized disease caused by the spread of bacteria in the blood (septicemia)—guided by bacterial culture and sensitivity testing of *E. coli*; possible antibiotics include amikacin, cefazolin, cefoxitin, ticarcillin-clavulanate



FOLLOW-UP CARE

PATIENT MONITORING

- Bacterial blood cultures—puppies/kittens with fever and/or diarrhea
- Monitor temperature—with signs of sluggishness (lethargy) and/or depression
- Monitor behavior—eating, drinking, and/or nursing; adequate weight gain

PREVENTIONS AND AVOIDANCE

- Bitch/queen—good health; vaccinated; good nutritional status
- Clean and disinfect birthing environment, as directed by your pet's veterinarian; clean bedding after birth frequently
- Ensure adequate colostrum (first milk) intake of all litter mates
- Separate bitch/queen with nursing litter from other dogs or cats
- Keep the number of animals low in kennel or cattery rooms
- Wash hands and change clothes and shoes after handling other dogs/cats and before dealing with newborns

POSSIBLE COMPLICATIONS

- Death of puppies or kittens

EXPECTED COURSE AND PROGNOSIS

- Newborns—life-threatening disease; prognosis often poor; newborn may succumb rapidly; quick treatment with supportive care essential for survival
- Adults—self-limiting disease with supportive care, depending on the degree of dehydration and existence of other diseases



KEY POINTS

- Newborns—life-threatening disease with poor prognosis

COLITIS AND PROCTITIS (INFLAMMATION OF THE COLON AND RECTUM)



BASICS

OVERVIEW

- “Colitis” is inflammation of the colon
- “Proctitis” is inflammation of the rectum

GENETICS

- Breed susceptibility to histiocytic ulcerative colitis in boxers; histiocytic ulcerative colitis is inflammation characterized by a thickened lining of the colon with varying degrees of loss of the superficial lining (known as “ulceration”); the thickening is due to infiltration of various cells (histiocytes, plasma cells, and lymphocytes) in the layers under the lining

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dogs—boxers for histiocytic ulcerative colitis; histiocytic ulcerative colitis is inflammation characterized by a thickened lining of the colon with varying degrees of loss of the superficial lining (known as “ulceration”); the thickening is due to infiltration of various cells (histiocytes, plasma cells, and lymphocytes) in the layers under the lining

Mean Age and Range

- Any age
- Boxers usually have clinical signs by 2 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Feces vary from semi-formed to liquid
- High frequency of defecation, with small volume of stool
- Animals often demonstrate prolonged straining (known as “tenesmus”) after defecation
- Long-term (chronic) diarrhea often with mucus and/or blood; cats may have formed feces with blood (known as “hematochezia”)
- Vomiting in approximately 30% of affected dogs
- Weight loss is rare
- Physical examination usually normal; dogs with histiocytic ulcerative colitis may show signs of weight loss and lack of appetite (known as “anorexia”); histiocytic ulcerative colitis is inflammation characterized by a thickened lining of the colon with varying degrees of loss of the superficial lining (known as “ulceration”); the thickening is due to infiltration of various cells (histiocytes, plasma cells, and lymphocytes) in the layers under the lining

CAUSES

- Infectious—parasites (such as whipworms [*Trichuris vulpis*], hookworms [*Ancylostoma caninum*], *Entamoeba histolytica*, *Balantidium coli*, *Giardia*, *Tritrichomonas*, *Cryptosporidium*), bacteria (such as *Salmonella*, *Clostridium*, *Campylobacter*, *Yersinia enterocolitica*, *Escherichia coli*), algae (*Prototheca*), fungus (*Histoplasma capsulatum*), and pythiosis/phycomycosis
- Trauma—foreign body, abrasive material
- Excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”)
- Segmental—secondary to long-term (chronic) inflammation of the pancreas (known as “pancreatitis”)
- Allergic—dietary protein, possibly bacteria
- Inflammatory/immune disorders—characterized by the type of cells found in the inflamed colon, such as lymphoplasmacytic, eosinophilic, granulomatous, and histiocytic colitis



TREATMENT

HEALTH CARE

- Outpatient medical management, unless diarrhea is severe enough to cause dehydration
- Balanced electrolyte fluids for dehydrated patients

DIET

- Patients with sudden (acute) inflammation of the colon (colitis) can be fasted for 24 to 48 hours
- Try a hypoallergenic or novel protein (a protein to which the animal has never been exposed) diet in patients with inflammatory

colitis; use a commercial or home-prepared diet that contains a protein to which the dog or cat has not been exposed

- Fiber supplementation with poorly fermented fiber (such as bran and α -cellulose) is recommended to increase fecal bulk, improve colonic muscle contractility, and bind fecal water to produce formed feces
- Some fermentable fiber (such as psyllium or a diet containing beet pulp or fructo-oligosaccharides) may be beneficial—short-chain fatty acids produced by fermentation may help the colon heal and restore normal colonic bacteria

SURGERY

- Segments of colon severely affected by scar tissue (known as “fibrosis”) from long-term (chronic) inflammation and subsequent narrowing (stricture formation) may need surgical removal; folding of one segment of the intestine into another segment (known as “intussusception”) requires surgical intervention; inflammation secondary to the water mold, *Pythium* (disease known as “pythiosis”) or to a particular fungal infection (known as “phycomycosis”) often requires surgical removal or debulking



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antiparasitic or Antimicrobial Drugs

- Whipworms (*Trichuris*), hookworms (*Ancylostoma*), and *Giardia*—fenbendazole (repeat treatment in 3 months)
- *Entamoeba*, *Balantidium*, and *Giardia*—metronidazole
- *Giardia*—albendazole may be an alternative medication to treat giardiasis, if fenbendazole or metronidazole is ineffective; monitor for bone-marrow suppression
- *Tritrichomonas foetus*—possibly ronidazole
- *Salmonella*—treatment is controversial because a carrier state can be induced; in patients with generalized (systemic) disease involvement, choose the antibiotic on the basis of bacterial culture and sensitivity testing (antibiotic examples include enrofloxacin, chloramphenicol, or trimethoprim-sulfa)
- *Clostridium*—metronidazole or tylosin
- *Campylobacter*—erythromycin or tylosin
- *Yersinia* and *E. coli*—choose the drug on the basis of bacterial culture and sensitivity testing
- *Prototheca*—no known treatment
- *Histoplasma*—itraconazole; amphotericin B in advanced cases
- Pythiosis/phycomycosis—amphotericin B lipid complex

Anti-Inflammatory and Immunosuppressive Drugs for Inflammatory/Immune Colitis

- Sulfasalazine; long-term use may be needed
- Steroids—prednisone (taper dosage slowly over 4 to 6 months as directed by your pet’s veterinarian, once clinical remission is achieved)
- Azathioprine—a chemotherapeutic drug used to decrease the immune response (dogs)
- Sulfasalazine or other 5-ASA drugs—may be the drugs of choice for plasmacytic lymphocytic colitis (inflammation of the colon characterized by the presence of plasma cells and lymphocytes; plasma cells are specialized white-blood cells; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease; a lymphocyte is a type of white-blood cell, formed in lymphatic tissue throughout the body)
- Prednisone and azathioprine are indicated in treatment of eosinophilic colitis (inflammation of the colon characterized by the presence of eosinophils; eosinophils are a type of white-blood cell; they are involved in allergic responses by the body and are active in fighting larvae of parasites) and severe plasmacytic lymphocytic colitis that does not respond to other therapies
- Histiocytic ulcerative colitis in dogs may respond to treatment with enrofloxacin alone or in combination with metronidazole and amoxicillin for 6 weeks; histiocytic ulcerative colitis is inflammation characterized by a thickened lining of the colon with varying degrees of loss of the superficial lining (ulceration); the thickening is due to infiltration of various cells (histiocytes, plasma cells, and lymphocytes) in the layers under the lining

Motility Modifiers (Symptomatic Relief Only)

- Loperamide
- Diphenoxylate
- Propantheline bromide, if colonic spasm is contributing to clinical signs



FOLLOW-UP CARE

PATIENT MONITORING

- Infrequent recheck examinations or client communication by phone

PREVENTIONS AND AVOIDANCE

- Avoid exposure to infectious agents (such as exposure to other dogs, contaminated foods, moist environments)

- Avoid sudden changes in diet

POSSIBLE COMPLICATIONS

- Recurrence of signs without treatment, when treatment is tapered, and with progression of disease
- Narrowing of the colon or rectum (stricture formation) due to long-term (chronic) inflammation

EXPECTED COURSE AND PROGNOSIS

- Most infections causes—excellent with treatment
- Infection with *Prototheca* (type of algae)—grave; no known treatment except surgical removal of diseased tissue
- Infection with *Histoplasma* (type of fungus)—poor in advanced or widespread (disseminated) disease; mild to moderate cases generally respond to therapy
- Pythiosis/phycomycosis—guarded to poor; poorly responsive to treatment; some dogs have fair results with surgical removal of affected tissue and treatment with amphotericin B lipid complex
- Traumatic, uremic, and segmental—good, if underlying cause is treatable
- Cecal inversion, ileoceocolic intussusception, and polyps—good with surgical removal
- Inflammatory—good with treatment in patients with lymphoplasmacytic, eosinophilic, and possibly histiocytic disease



KEY POINTS

- Treatment may be intermittent and long-term in patients with inflammatory/immune colitis, and repeated recurrence is seen in some cases
- Some types of colitis respond poorly to medical treatment; surgery may be necessary

COMPULSIVE DISORDERS—CATS



BASICS

OVERVIEW

- Repetitive, relatively constant, exaggerated behavior patterns with no apparent reason or function
- Behaviors such as psychological hair loss in which the cat grooms excessively (known as “psychogenic alopecia”), compulsive pacing, frequently repeated meowing or vocalizing, and sucking or fabric chewing may be considered compulsive disorders, when other reasons for the behavior cannot be identified

SIGNALMENT/DESCRIPTION of ANIMAL

- Any age, sex, or breed of cat may have compulsive disorders
- Siamese, other Asian breeds and Asian-breed crosses may be more likely to demonstrate repeated vocalization and fabric chewing

SIGNS/OBSERVED CHANGES in the ANIMAL

Signs or observed changes are determined by the abnormal behavior itself. A cat may demonstrate one or more abnormal behaviors.

- Psychogenic alopecia—localized, symmetrical hair loss; most commonly involving the skin of the groin, lower abdomen, and inner thigh or back of thighs; appearance of the skin may be normal or may be abnormal, with redness or abrasions from excessive grooming
- Compulsive pacing
- Repeated meowing or vocalizing
- Fabric sucking/chewing—secondary gastrointestinal signs, such as vomiting, may develop

Once started, these behaviors may quickly increase in frequency if they are reinforced in some way, such as the owner feeding or giving attention to the cat while it is exhibiting the abnormal behavior

CAUSES

- Unknown
- Organic or physical causes for the abnormal behavior should be ruled out before a psychological basis is presumed

RISK FACTORS

- Changes in surroundings may lead to abnormal behaviors
- More commonly reported in indoor cats; may be an artifact of the higher level of attention such pets receive or may be related to the stress of confinement or social isolation similar to the pacing and other forms of barrier frustration seen in large cats in zoos



TREATMENT

HEALTH CARE

- Reduce environmental stress—increase the predictability of household events by establishing a consistent schedule for feeding, playing, exercise, and social time; eliminate unpredictable events as much as possible; avoid confinement
- Psychogenic alopecia—topical agents to discourage licking/grooming are usually ineffective
- Compulsive pacing—allowing the cat to go outside after the start of this behavior may reinforce it; if possible, let the cat out before the behavior begins
- Repetitive vocalizations—breed or spay an intact female; castrate an intact male
- Fabric chewing/sucking—keep fabrics of interest out of the cat’s reach; increase dietary roughage
- Do not reward the behavior
- Ignore the behavior as much as possible
- Note details of the time, place, and social environment so that an alternative positive behavior (such as play or feeding) may be scheduled
- Any punishment for an unwanted behavior associated with the owner’s voice, movement, and touch may increase the unpredictability of the cat’s environment, may increase the cat’s fear or aggressive behavior, and may disrupt the human/animal bond.

ACTIVITY

- Playing or exercise on a consistent schedule

DIET

- Increase dietary roughage for cats with fabric chewing/sucking behaviors

SURGERY

- Spay or neuter intact cats for repetitive vocalization



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Environmental control—preferred method of management; psychologically active drugs may be needed as well
- No drugs are approved by the FDA for the treatment of these disorders in cats; your veterinarian will discuss the risks and benefits of using these drugs
- Goal—use drugs until control is achieved for 2 months; then attempt gradual decrease in dosage
- Tricyclic antidepressant—amitriptyline or clomipramine
- Selective serotonin re-uptake inhibitor---fluoxetine
- Phenobarbital
- Deprenyl (selegiline), if signs related to cognitive dysfunction (changes in behavior and awareness related to aging of the brain)



FOLLOW-UP CARE

PATIENT MONITORING

- Before initiating treatment, record the frequency of abnormal behavior that occur each week so that progress can be monitored
- Successful treatment requires a schedule of follow-up examinations
- Environmental modification program and/or psychologically active medications must be adjusted according to the cat's response
- If a medication is not effective after dosage adjustment, another drug may be prescribed

PREVENTIONS AND AVOIDANCE

- Do not reward the behavior
- Ignore the behavior as much as possible

POSSIBLE COMPLICATIONS

- Fabric chewing/sucking—gastrointestinal problems, such as vomiting or blockage
- Irritation or frustration of people in household

EXPECTED COURSE AND PROGNOSIS

- Realistic expectations must be understood; changing a behavior that has become a habit is very challenging
- Immediate control of a long-standing problem is unlikely



KEY POINTS

- Any cat may develop repetitive, exaggerated behavior patterns that apparently have no reason or function
- Ignore the behavior as much as possible and avoid rewarding the behavior
- Abnormal behavior should be evaluated by your veterinarian as soon as possible to determine if a physical cause exists
- Treatment may include behavioral modification and psychologically active drugs if no physical cause is identified

OBSESSIVE-COMPULSIVE DISORDERS IN DOGS



BASICS

OVERVIEW

- Repetitious, relatively unchanging sequence of activities or movements that has no obvious purpose or function, usually derived from normal maintenance behaviors (such as grooming, eating, walking); the repetitive behavior interferes with normal behavioral functioning
- Called “OCD” or “Obsessive-Compulsive Disorder”
- Most common obsessive-compulsive signs are spinning; tail chasing; self-mutilation; hallucinating (“fly biting”); circling; fence running; hair/air biting; pica (appetite for non-food substances such as dirt, rocks); pacing; staring and vocalizing; self-directed vocalization; potentially some aggressions

SIGNALMENT/DESCRIPTION of ANIMAL

- No breed, sex or age of dog is more likely to have obsessive-compulsive disorders, although the type of OCD (such as spinning compared to self-mutilation) displayed by the dog may be affected by breed
- Signs begin early (at 12 to 24 months of age) in development of social maturity (generally defined as occurring at 12 to 36 months of age in dogs) like other anxiety disorders; have been seen in younger animals
- Bull terriers—tail chasing not uncommon and seems to run in families
- German shepherd dogs—spinning and tail chasing have been reported to be more common than in other breeds
- Great Danes and German short-haired pointers—some lines display self-mutilation, stereotypic motor behavior (such as fence running), or hallucinations; may be more familial than breed-associated, with incidence varying by region and not well documented
- Breed versus familial association is confusing in dogs; not all family members show the same manifestation (for example, spinning, grooming, or hallucinating) and, in fact, the opposite may be true; if the client sees signs developing in a dog from a line where other dogs are affected, early intervention is critical; treat all nonspecific ritualistic behaviors with increased exercise, behavior modification, and, minimally, nonspecific tricyclic antidepressants (TCAs)
- Obsessive component may be at core of the problem, although it is difficult to verify; only the resulting ritualistic behaviors are recognized easily

SIGNS/OBSERVED CHANGES in the ANIMAL

- May be nonspecific; physical examination findings may be normal or may be abnormal secondary to OCD behavior (for example, signs of self mutilation)
- The behavior may be a manifestation of OCD, if the client cannot interrupt it and if it intensifies over time, increases in frequency or duration, and interferes with normal functioning
- Videotape dogs in all circumstances where the client sees the behavior; a pattern may become clear
- Dog may have begun to chase its tail as part of play, but tail chasing became more frequent and now the tip is missing and even physical restraint does not stop the behavior (however, not all dogs that tail chase will mutilate their tails)
- May be seen in young dogs, but its onset is more common during social maturity; play decreases with age, OCD increases.
- A solitary focus may have seemed to spur the behavior (for example, chasing a mouse that the patient could not catch), but usually no provocative stimulus is noted
- Behavior worsens with time
- May see self-induced injuries and lack of condition that may be associated with increased motor activity and repetitive behaviors; may note self-mutilation with a focus on the tail, forelimbs, and distal extremities

CAUSES

- Illness or painful physical condition may increase a dog’s anxieties and contribute to these problems; few of these conditions actually cause OCD
- Kenneling and confinement may be associated with spinning
- Degenerative (for example, aging and related nervous-system changes), anatomic, infectious (primarily central nervous system [CNS] viral conditions), and toxic (for example, lead poisoning) causes may lead to signs, but abnormal behavior likely is rooted in primary or secondary abnormal nervous system chemical activity

RISK FACTORS

- Illness or painful physical condition may increase a dog’s anxieties
- Kenneling and confinement



TREATMENT

HEALTH CARE

- Most patients respond to a combination of behavior modification and anti-anxiety medication
- Anti-anxiety medication—implement early; may be a prerequisite to effecting any behavioral therapy
- Usually outpatient
- Inpatient—patients with severe self-mutilation and self-induced injury; patients that must be protected from the environment until the anti-anxiety medications reach effective levels (may require days to weeks of therapy); constant monitoring, stimulation, and care
- Sedation may be necessary in severe cases; only a stop-gap measure, but necessary if serious and acute mutilation involved
- Behavior modification—geared toward teaching the patient to relax in a variety of environmental settings and to substitute a calm, competitive or desired behavior for the obsessive-compulsive one
- Desensitization and counterconditioning—most effective if instituted early; may be coupled to a verbal cue that signals the patient to execute a behavior that is competitive with the abnormal one (for example, instead of circling, the patient is taught to relax and lie down with its head and neck stretched prone on the floor when the client says, “Head down”)
- Punishment should be avoided; may make the behavior worse and lead to the patient being more secretive
- The veterinarian should diagnose and control itchy skin diseases and painful conditions, as itchiness (known as “pruritus”) and pain are related to anxiety
- Confinement or physical restraint—avoid bandages, collars, braces, and crates; all serve to focus animal more on the center of its distress and will make the dog worse; if these are needed to ensure healing, they should be used for the minimum amount of time

ACTIVITY

- Increase exercise

DIET

- Dietary management to control some forms of itchy skin disease



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- **Tricyclic antidepressants (TCAs)** and selective serotonin reuptake inhibitors (SSRIs) **increase serotonin levels in the central nervous system**
- **Mild signs**—amitriptyline; imipramine; only useful for nonspecific ritualistic behaviors that may be associated with OCD
- **Severe or long-standing signs**—clomipramine; fluoxetine; these drugs may take 3–5 weeks to be effective
- May take months to get real effect
- Self-mutilation—narcotic antagonists (such as naltrexone) may be useful, but expensive and have a short effective period in the dog
- Thioridazine—occasionally used as an adjuvant treatment; newer more specific treatments appear more effective; some antipsychotic drugs (such as risperidone, olanzapine, clozapine)
- Treatment is lifelong; any attempt to withdraw medication should be gradual; recurrence is common



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor behaviors via weekly videotaping and written logs; will provide unbiased assessments of change and help with alterations in treatment plans
- Complete blood count, biochemistry profile and urinalysis—semiannually or yearly if the patient is on chronic treatment; adjust dosages accordingly
- Observe for vomiting, gastrointestinal distress, and rapid breathing (tachypnea); if these signs are identified, contact your pet’s veterinarian
- Medications may take several weeks to show an effect on the target behavior—the first sign of efficacy may be change in the duration or frequency of bouts rather than total cessation of the undesired behaviors
- Setting realistic expectations for change will help owners manage the pet and the outcome of behavioral and medical intervention
- Relapses can be common during stressful situations

PREVENTIONS AND AVOIDANCE

- Discourage the client from reassuring the patient that it does not have to spin, chew, or perform other repetitive behaviors; this inadvertently rewards the repetitive behavior; have them reward dog only when not engaged in behavior and relaxed

EXPECTED COURSE AND PROGNOSIS

- If left untreated, these conditions almost always progress



KEY POINTS

- Repetitious, relatively unchanging sequence of activities or movements that has no obvious purpose or function, usually derived from normal maintenance behaviors (such as grooming, eating, walking); the repetitive behavior interferes with normal behavioral functioning
- Most patients respond to a combination of behavior modification and anti-anxiety medication
- Monitor behaviors via weekly videotaping and written logs; will provide unbiased assessments of change and help with alterations in treatment plans
- If left untreated, these conditions almost always progress

LEFT-SIDED CONGESTIVE HEART FAILURE



BASICS

OVERVIEW

- Failure of the left side of the heart to pump blood at a sufficient rate to meet the needs of the body or to prevent blood from pooling within the veins of the lungs
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- The “left side” of the heart consists of the left atrium and the left ventricle

GENETICS

- Some congenital (present at birth) heart defects, heart-muscle diseases (known as “cardiomyopathies”), and heart valve diseases have a genetic basis in some breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Vary with cause

Mean Age and Range

- Vary with cause

Predominant Sex

- Varies with cause

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs vary with underlying cause
- Signs vary with the species involved
- Weakness, sluggishness (lethargy), exercise intolerance
- Coughing (dogs) and difficulty breathing (known as “dyspnea”); breathing signs often worsen at night and can be relieved by assuming a standing position, lying on the chest bone (known as “sternal recumbency”), or standing with the elbows away from the body in an attempt to increase lung capacity (known as “orthopnea”)
- Cats rarely cough from heart failure
- Rapid breathing (known as “tachypnea”)
- Coughing, often soft in conjunction with rapid breathing (tachypnea) in dogs
- Abnormal breath sounds on listening to the lungs with a stethoscope (known as “auscultation”)—short, rough snapping sounds (known as “crackles”); and squeaking or whistling sounds (known as “wheezes”)
- Pale/gray/bluish (known as “cyanotic”) gums and moist tissues of the mouth (known as “mucous membranes”)
- Pink color of the gums is slow to return when the gums are blanched by finger pressure (known as “prolonged capillary refill time”)
- Possible heart murmur or gallop rhythm heard when listening to the heart with a stethoscope
- Weak femoral pulses

CAUSES

Pump (Muscle) Failure of the Left Ventricle

- Heart muscle is flabby and weak for unknown causes (so called “idiopathic dilated cardiomyopathy” or “DCM”)
- Trypanosomiasis (disease caused by the protozoa, *Trypanosoma*)—rare
- Toxicity of doxorubicin (a chemotherapeutic drug) to the heart in dogs
- Inadequate levels of thyroid hormone (known as “hypothyroidism”)—rare
- Excessive levels of thyroid hormone (known as “hyperthyroidism”)—rarely causes pump failure

Pressure Overload of the Left Side of the Heart

- Generalized (systemic) high blood pressure (known as “hypertension”)
- Birth defect involving narrowing just below the aortic valve, the heart valve from the left ventricle to the aorta (the main artery of the body; condition known as “subaortic stenosis”)
- Narrowing of the aorta (known as “coarctation of the aorta”)—rare; Airedale terriers more likely than other breeds to have this condition
- Tumors of the left ventricle—rare

Volume Overload of the Left Side of the Heart

- Long-term (chronic) mitral valve disease (known as “endocardiosis”)—dogs; the “mitral valve” is the heart valve between the left atrium and the left ventricle
- Abnormal development of the mitral valve (known as “mitral valve dysplasia”)—cats and dogs; the “mitral valve” is the heart valve

between the left atrium and the left ventricle

- Developmental abnormalities or birth defects involving the heart (such as patent ductus arteriosus or PDA in dogs and ventricular septal defect in dogs and cats)
- Backward flow of blood through the aortic valve (known as “aortic valve insufficiency”) secondary to infection/inflammation of the lining of the heart (known as “endocarditis”)—dogs; the “aortic valve” is the valve from the left ventricle to the aorta (the main artery of the body)

Impediment to Filling of the Left Side of the Heart

- Fluid build-up between the heart and the sac surrounding the heart (known as “pericardial effusion”) with resulting compression of the heart (known as “tamponade”)
- Inflammation of the sac surrounding the heart (pericardium) with thickening and scarring, such that it restricts filling of the heart with blood as the ventricles cannot expand normally (known as “constrictive pericarditis” or “restrictive pericarditis”)
- Heart-muscle disease in which the muscle is “stiff” and does not expand, such that blood cannot fill the ventricles normally (known as “restrictive cardiomyopathy”)
- Disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle (known as “hypertrophic cardiomyopathy” or “HCM”)
- Masses (such as a tumor or blood clot [known as a “thrombus”]) in the left atrium
- Blood clots in the lungs (known as “pulmonary thromboembolism”)
- Narrowing of the mitral valve (known as “mitral stenosis”)—rare; the “mitral valve” is the heart valve between the left atrium and the left ventricle

Heart Rate or Rhythm Disturbances

- Slow heart rate (known as “bradycardia”)
- Rapid heart rate (known as “tachycardia”)

RISK FACTORS

- Conditions causing long-term (chronic), high blood volume being pumped by the heart (known as “high cardiac output”), such as excessive levels of thyroid hormone (hyperthyroidism), low red-blood cell count (known as “anemia”), and pregnancy



TREATMENT

HEALTH CARE

- Usually treat as outpatient, unless animal is having difficulty breathing (dyspnea) or has very low blood pressure (known as “severe hypotension”)
- Identify and correct underlying cause whenever possible
- Minimize handling of animals that are having critical breathing difficulties (critical dyspnea)—stress can kill!
- Oxygen is life saving in animals that are having critical breathing difficulties (critical dyspnea)

ACTIVITY

- Restrict activity

DIET

- Initiate moderately sodium-restricted diet
- Severe sodium restriction is indicated in animals with advanced disease

SURGERY

- Surgery may benefit selected patients with congenital (present at birth) defects, such as patent ductus arteriosus (PDA), and some forms of heart valve disease; response to surgery varies
- Tapping and draining the space between the heart and the sac surrounding the heart (pericardium; procedure known as “pericardiocentesis”) in animals with fluid build-up (pericardial effusion)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Medications to Remove Excess Fluid from the Body (known as “Diuretics”)

- Furosemide or another “loop diuretic” is the initial diuretic of choice; diuretics are indicated to remove fluid build-up from the lungs (known as “pulmonary edema”); animals that are having critical breathing difficulties (critical dyspnea) often require high doses given intravenously to stabilize them; once the fluid build-up in the lungs resolves, the dose gradually should be reduced to the lowest effective dosage, as directed by your pet’s veterinarian

- Spironolactone increases survival in people with heart failure because of its ability to block aldosterone (a hormone produced by the adrenal gland; involved in sodium and potassium regulation in the body); use in combination with furosemide
- Thiazide diuretics can be added to furosemide and spironolactone in heart failure cases that do not respond to these medications

Digoxin (Type of Heart Medication)

- Digoxin is used in animals with heart-muscle failure (such as dilated cardiomyopathy, a disease characterized by a flabby, weak heart muscle)
- Digoxin also is indicated to treat certain abnormal or irregular heart rhythms that start in heart tissue above the ventricles (known as “supraventricular arrhythmias”) in patients with congestive heart failure
- In people, digoxin has no effect on mortality, but decreases hospitalization due to heart failure

Medications to Enlarge or Dilate the Veins (known as “Venodilators”)

- Nitroglycerin ointment causes dilation of the veins, thus lowering the pressure required to fill the left atrium
- Used for immediate stabilization of patients with severe fluid build-up in their lungs (pulmonary edema) and difficulty breathing (dyspnea)
- May be useful in animals with long-term (chronic) left-sided congestive heart failure; to avoid tolerance to the drug, use intermittently and with 12-hour dose-free interval between the last dose of one day and the first dose of the next day

Angiotensin-Converting Enzyme (ACE) Inhibitors (Type of Heart Medication)

- Angiotensin-converting enzyme (ACE) inhibitor, such as enalapril or benazepril, is indicated in most animals with left-sided congestive heart failure
- ACE inhibitors improve survival and quality of life in dogs with left-sided congestive heart failure secondary to degenerative valve disease and dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle)

Medications that Improve Heart-Muscle Contraction (known as “Positive Inotropes”)

- Dobutamine is a potent medication that improves heart-muscle contraction (positive inotropic agent) and may provide valuable short-term support of a heart failure patient with poor heart-muscle contractility
- Pimobendan is a calcium-channel sensitizer that dilates arteries and increases heart-muscle contraction—first line medication in treating dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle); useful in dogs with congestive heart failure due to long-term (chronic) heart valve disease that does not respond to medical treatment
- Positive inotropes can cause irregular heart beats; careful monitoring is required

Medications to Control Irregular Heart Beats (known as “Antiarrhythmic Agents”)

- Treat irregular heart beats (known as “arrhythmias”), as needed

Medications to Enlarge or Dilate Arteries (known as “Arterial Dilators”)

- Hydralazine or amlodipine can be substituted for an angiotensin-converting enzyme (ACE) inhibitor in patients that do not tolerate the drug or have advanced kidney failure; monitor for low blood pressure (hypotension) and rapid heart rate (tachycardia); can be added to an angiotensin-converting enzyme (ACE) inhibitor cautiously in animal with left-sided congestive heart failure that does not respond to medical treatment
- Nitroprusside is a potent medication that dilates arteries (arterial dilator); it usually is reserved for short-term support of patients with life-threatening fluid build-up in their lungs (pulmonary edema)

Calcium Channel Blockers

- Diltiazem frequently is used in patients with left-sided congestive heart failure for heart rate control in animals with abnormal or irregular heart rhythms that start in heart tissue above the ventricles (supraventricular arrhythmias) not controlled by digoxin and in cats with hypertrophic cardiomyopathy (disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle)

Beta Blockers

- Atenolol and metoprolol are used for heart rate control in animals with rapid heart rhythms that start in heart tissue above the ventricles (known as “supraventricular tachycardia”), hypertrophic cardiomyopathy (disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle), and excessive levels of thyroid hormone (hyperthyroidism)
- Used alone or with a medication to control irregular ventricular heart beats (antiarrhythmic drug); these drugs depress heart-muscle contraction (known as “negative inotropes”), so they should be used cautiously in patients with heart-muscle failure
- On basis of human studies, may enhance survival in animals with idiopathic dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle of unknown cause)

Nutritional Supplements

- Potassium supplementation, if low levels of potassium in the blood (known as “hypokalemia”) are documented; use potassium supplements cautiously in animals receiving an angiotensin-converting enzyme (ACE) inhibitor or spironolactone
- Taurine supplementation in cats with dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle) and dogs with dilated cardiomyopathy and taurine deficiency (such as American cocker spaniels); taurine is an amino acid (protein) that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the heart; dogs may be affected by inadequate levels of taurine as well
- L-carnitine supplementation may help some dogs with dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle)
- Coenzyme Q₁₀ is of potential value based on the results of small trials in people with dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle)



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor kidney status, electrolytes, hydration, breathing rate and effort, heart rate, body weight, and abdominal girth (dogs)
- If excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) develop, reduce the dosage of diuretic, as directed by your pet’s veterinarian; if azotemia persists and the animal also is on an angiotensin-converting enzyme (ACE) inhibitor, reduce or discontinue the ACE inhibitor, as directed by your pet’s veterinarian
- Use digoxin with caution if excess levels of urea and other nitrogenous waste products in the blood (azotemia) develop
- Monitor electrocardiogram (“ECG,” a recording of the electrical activity of the heart) if irregular heart beats (arrhythmias) are suspected
- Check digoxin concentration in the blood periodically

PREVENTIONS AND AVOIDANCE

- Minimize stress, exercise, and sodium intake in patients with heart disease
- Use of an angiotensin-converting enzyme (ACE) inhibitor early in the course of heart disease in patients with dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle) may slow the progression of heart disease and delay onset of congestive heart failure
- Use of an angiotensin-converting enzyme (ACE) inhibitor in animals with mitral valve disease that are not showing signs of disease remains controversial

POSSIBLE COMPLICATIONS

- Fainting (known as “syncope”)
- Blood clots in the aorta, the main artery of the body (known as “aortic thromboembolism”) in cats
- Irregular heart beats (arrhythmias)
- Electrolyte imbalances
- Digoxin toxicity
- Excess levels of urea and other nitrogenous waste products in the blood (azotemia) and kidney failure

EXPECTED COURSE AND PROGNOSIS

- Prognosis varies with underlying cause



KEY POINTS

- Failure of the left side of the heart to pump blood at a sufficient rate to meet the needs of the body or to prevent blood from pooling within the veins of the lungs
- Some congenital (present at birth) heart defects, heart-muscle diseases (known as “cardiomyopathies”), and heart valve diseases have a genetic basis in some breeds
- Left-sided congestive heart failure is not curable, with few exceptions (such as animals with thyroid disorders, irregular heart beats [arrhythmias], nutritionally responsive heart disease)

RIGHT-SIDED CONGESTIVE HEART FAILURE



BASICS

OVERVIEW

- Failure of the right side of the heart to pump blood at a sufficient rate to meet the needs of the body or to prevent blood from pooling within the veins of the body
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- The “right side” of the heart consists of the right atrium and the right ventricle

GENETICS

- Some congenital (present at birth) heart defects have a genetic basis in certain breeds
- A heart-muscle disease of the right ventricle accompanied by irregular heart beats (known as “arrhythmogenic right ventricular cardiomyopathy” or “ARVC”) appears to have a genetic basis in boxers

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Vary with cause

Mean Age and Range

- Vary with cause

Predominant Sex

- Varies with cause

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs vary with underlying cause
- Signs vary with the species involved
- Weakness
- Sluggishness (lethargy)
- Exercise intolerance
- Abdominal enlargement or distension
- Difficulty breathing (known as “dyspnea”), rapid breathing (known as “tachypnea”)
- Enlargement or distention of the jugular vein; the jugular veins are located on either side of the neck; they are the main blood vessels through which blood is returned from the head to the heart
- Pulsation may be detected in the jugular veins (known as a “jugular pulse”) in some animals
- Enlarged liver (known as “hepatomegaly”)
- Fluid build-up in the abdomen (known as “ascites”) is common in dogs and rare in cats with right-sided congestive heart failure
- Fluid build-up in the space between the lungs and chest wall (known as “pleural effusion”) without fluid build-up in the abdomen (ascites) and liver enlargement (hepatomegaly) is rare in dogs with right-sided congestive heart failure
- Fluid build-up in the abdomen (ascites) without fluid build-up in the space between the lungs and chest wall (pleural effusion) is rare in cats with right-sided congestive heart failure
- Possible heart murmur may be heard when listening to the heart with a stethoscope
- Muffled heart sounds may be heard when listening to the heart with a stethoscope, if animal has fluid build-up in the space between the lungs and chest wall (pleural effusion) or in the space between the heart and the sac surrounding the heart (the sac is the “pericardium;” condition known as “pericardial effusion”)
- Weak femoral pulses
- Rapid, shallow breathing, if animal has fluid build-up in the space between the lungs and chest wall (pleural effusion) or severe fluid build-up in the abdomen (ascites)
- Fluid build-up in the tissues, especially the legs and under the skin (known as “peripheral edema”) is infrequent

CAUSES

Pump (Myocardial) Failure of the Right Ventricle

- Heart muscle is flabby and weak for unknown causes (so called “idiopathic dilated cardiomyopathy” or “DCM”)
- A heart-muscle disease of the right ventricle accompanied by irregular heart beats (arrhythmogenic right ventricular cardiomyopathy or ARVC)
- Trypanosomiasis (disease caused by the protozoa, *Trypanosoma*)
- Toxicity of doxorubicin (a chemotherapeutic drug) to the heart
- Long-term (chronic) excessive levels of thyroid hormone (known as “hyperthyroidism”)

Volume Overload of the Right Ventricle

- Long-term (chronic) mitral and/or tricuspid valve disease (known as “endocardiosis”); the “mitral valve” is the heart valve between the left atrium and the left ventricle; the “tricuspid valve” is the heart valve between the right atrium and the right ventricle
- Abnormal development of the tricuspid valve (known as “tricuspid valve dysplasia”); the “tricuspid valve” is the heart valve between the right atrium and the right ventricle

Pressure Overload of the Right Ventricle

- Heartworm disease
- Long-term (chronic) obstructive lung disease, with high blood pressure in the lungs (known as “pulmonary hypertension”)
- Blood clots to the lungs (known as “pulmonary thromboembolism”)
- Narrowing of the pulmonary valve (known as “pulmonic stenosis”); the “pulmonary valve” is the heart valve from the right ventricle to the main pulmonary (lung) artery
- Tetralogy of Fallot, a set of birth defects in the heart
- Tumor in the right ventricle
- Primary high blood pressure in the lungs (pulmonary hypertension)

Impediment to Blood Filling of the Right Ventricle

- Fluid build-up between the heart and the sac surrounding the heart (pericardium; condition is pericardial effusion) with resulting compression of the heart (known as “tamponade”)
- Inflammation of the sac surrounding the heart (pericardium) with thickening and scarring, such that it restricts filling of the heart with blood as the ventricles cannot expand normally (known as “constrictive pericarditis” or “restrictive pericarditis”)
- Tumors or masses in the right atrium or vena cava; the “vena cava” is the main vein that returns blood from the body to the heart
- Narrowing of the tricuspid valve (known as “tricuspid stenosis”); the “tricuspid valve” is the heart valve between the right atrium and the right ventricle
- Cor triatriatum dexter, a birth defect of the heart

Heart Rate or Rhythm Disturbances

- Slow heart rate (known as “bradycardia”), generally due to a complete heart block
- Rapid heart rate (known as “tachycardia”) and/or rapid, irregular heart beats (known as “tachyarrhythmias”)

RISK FACTORS

- No heartworm preventive medication or lack of routine administration of heartworm preventive medication
- Offspring of animal with right-sided congenital (present at birth) heart defect
- Conditions causing long-term (chronic), high blood volume being pumped by the heart (known as “high cardiac output”), such as excessive levels of thyroid hormone (hyperthyroidism), low red-blood cell count (known as “anemia”), and pregnancy



TREATMENT

HEALTH CARE

- Most animals treated as outpatients, unless having difficulty breathing (dyspnea) or collapse
- Tapping the chest to remove excess fluid (known as “thoracentesis”) and tapping the abdomen to remove excess fluid (known as “abdominocentesis”) may be required periodically for patients no longer responsive to medical management or for those with severe difficulty breathing (dyspnea) due to fluid build-up in the space between the lungs and chest wall (pleural effusion) or in the abdomen (ascites)

ACTIVITY

- Restrict activity

DIET

- Restrict sodium moderately
- Severe sodium restriction is indicated for animals with advanced disease

SURGERY

- Surgery may benefit selected patients with congenital (present at birth) defects, such as those with narrowing of the pulmonary valve (known as “pulmonic stenosis”); the “pulmonary valve” is the heart valve from the right ventricle to the main pulmonary (lung) artery
- Tapping and draining the space between the heart and the sac surrounding the heart (pericardium; procedure known as “pericardiocentesis”) or surgically removing the pericardium (known as “pericardectomy”) in animals with fluid build-up (pericardial effusion)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drugs should be administered only after a definitive diagnosis is made

Medications to Remove Excess Fluid from the Body (known as “Diuretics”)

- Furosemide or another “loop diuretic” is the initial diuretic of choice; diuretics are indicated to remove fluid build-up
- Spironolactone increases survival in people with heart failure because of its ability to block aldosterone (a hormone produced by the adrenal gland; involved in sodium and potassium regulation in the body); use in combination with furosemide
- Thiazide diuretics can be added to furosemide and spironolactone in heart failure cases that do not respond to these medications

Digoxin

- Digoxin is used in animals with heart-muscle failure (such as dilated cardiomyopathy, a disease characterized by a flabby, weak heart muscle)
- Digoxin also is indicated to treat certain abnormal or irregular heart rhythms that start in heart tissue above the ventricles (known as “supraventricular arrhythmias”) in patients with congestive heart failure

Angiotensin-Converting Enzyme (ACE) Inhibitors (Type of Heart Medication)

- Angiotensin-converting enzyme (ACE) inhibitor, such as enalapril or benazepril, is helpful in dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle) and long-term (chronic) mitral and/or tricuspid valve disease; the “mitral valve” is the heart valve between the left atrium and the left ventricle; the “tricuspid valve” is the heart valve between the right atrium and the right ventricle

Pimobendan

- Calcium sensitizer that enlarges or dilates arteries (known as “arterial vasodilation”) and increases heart-muscle contraction
- Especially useful in heart-muscle failure

Other Medications

- Potassium supplementation, if low levels of potassium in the blood (known as “hypokalemia”) are documented; use potassium supplements cautiously in animals receiving an angiotensin-converting enzyme (ACE) inhibitor or spironolactone
- Treat irregular heart beats (known as “arrhythmias”), as needed
- Taurine supplementation in cats with dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle) and dogs with dilated cardiomyopathy and taurine deficiency (such as American cocker spaniels); taurine is an amino acid (protein) that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the heart; dogs may be affected by inadequate levels of taurine as well
- L-carnitine supplementation may help some dogs with dilated cardiomyopathy (disease characterized by a flabby, weak heart muscle), such as in cocker spaniels and boxers



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor kidney status, electrolytes, hydration, breathing rate and effort, body weight, and abdominal girth (dogs)
- If excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) develop, reduce the dosage of diuretic, as directed by your pet’s veterinarian; if azotemia persists and the animal also is on an angiotensin-converting enzyme (ACE) inhibitor, reduce or discontinue the ACE inhibitor, as directed by your pet’s veterinarian
- Use digoxin with caution if excess levels of urea and other nitrogenous waste products in the blood (azotemia) develop
- Monitor electrocardiogram (“ECG,” a recording of the electrical activity of the heart) if irregular heart beats (arrhythmias) are suspected
- Check digoxin concentration in the blood periodically

POSSIBLE COMPLICATIONS

- Blood clots in the lungs (pulmonary thromboembolism)
- Irregular heart beats (arrhythmias)
- Electrolyte imbalances
- Digoxin toxicity
- Excess levels of urea and other nitrogenous waste products in the blood (azotemia) and kidney failure

EXPECTED COURSE AND PROGNOSIS

- Prognosis varies with underlying cause



KEY POINTS

- Failure of the right side of the heart to pump blood at a sufficient rate to meet the needs of the body or to prevent blood from pooling within the veins of the body
- Some congenital (present at birth) heart defects have a genetic basis in certain breeds
- Right-sided congestive heart failure is not curable, with few exceptions (such as animals with heartworm disease, irregular heart beats [arrhythmias], excessive levels of thyroid hormone [hyperthyroidism] and fluid build-up in the space between the heart and the sac that

surrounds the heart for unknown cause [so called “idiopathic pericardial effusion”])

- Most patients improve with initial treatment, but often have recurrent heart failure

INFLAMMATION OF THE MOIST TISSUES OF THE EYE (CONJUNCTIVITIS) IN CATS



BASICS

OVERVIEW

- Inflammation of the moist tissues of the eye (known as the “conjunctiva”); the conjunctiva is the vascularized moist tissue (mucous membrane) that covers the front part of the eyeball or globe, up to the edge of the cornea (known as the “bulbar conjunctiva”) and lines the lids and third eyelid (known as the “palpebral conjunctiva”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Breed Predispositions

- Infectious inflammation of the moist tissues of the eye (conjunctivitis)—purebred cats seem to be more likely to develop infections involving the conjunctiva than other cats

Mean Age and Range

- Infections—most commonly affect young animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Squinting or spasmodic blinking (known as “blepharospasm”)
- Redness of the moist tissues of the eye (known as “conjunctival hyperemia”)
- Discharge from the eye(s); may be clear or may contain mucus and/or pus
- Fluid build-up (known as “edema”) of the moist tissue covering of the eyeball (bulbar conjunctiva), around the cornea (condition known as “chemosis”)
- Upper respiratory infection—possible

CAUSES

Viral Causes

- Feline herpesvirus (FHV)—most common infectious cause; only one that leads to corneal changes (such as corneal ulcers)
- Calicivirus

Bacterial Causes

- Primary condition (that is, not secondary to another condition such as “dry eye” [known as “keratoconjunctivitis sicca” or “KCS”])—rare, except for *Chlamydia* and *Mycoplasma* infections
- Newborn inflammation of the moist tissues of the eye (conjunctivitis)—accumulation of discharge, often associated with a bacterial or viral infection; seen before the eyelids separate or open

Immune-mediated Causes

- Eosinophilic inflammation; “eosinophils” are a type of white-blood cell; they are involved in allergic responses by the body
- Related to generalized (systemic) immune-mediated diseases—such as pemphigus, in which the body attacks its own tissues

Cancer

- Rare cause of inflammation of the moist tissues of the eye (conjunctivitis); lymphoma and squamous cell carcinoma are the most common type of cancer that affect the conjunctiva

Secondary to Disease of the Tissues Surrounding the Eye (known as “adnexa,” such as eyelids, third eyelid, and tear glands)

- Lack of normal tear film (known as “aqueous tear film deficiency”)
- May develop “dry eye” (KCS) as a result of scarring
- Lid diseases (such as “entropion,” in which the eyelid curls inward, allowing facial hair to rub the eye)—may lead to clinical signs of inflammation of the moist tissues of the eye (conjunctivitis)
- Secondary to blockage of the outflow portion of the drainage system that normally moves tears to the nasal passages (known as the “nasolacrimal system”), such as an obstructed nasolacrimal duct

Secondary to Trauma or Environmental Causes

- Foreign body located in the moist tissues of the eye
- Irritation from dust, chemicals, or eye medications

Secondary to Other Eye Diseases

- Disorder of the cornea (the clear outer layer of the front of the eye) characterized by the presence of ulcers, with or without inflammation (condition known as “ulcerative keratitis”)
- Inflammation of the front part of the eye, including the iris (known as “anterior uveitis”)
- Disease of the eye, in which the pressure within the eye is increased (known as “glaucoma”)

RISK FACTORS

- Exposure to cats with active viral infections (such as feline herpesvirus) or carrying the virus or with bacterial infections (such as *Chlamydia*)
- Stress in cats with history of feline herpesvirus (FHV) infection



TREATMENT

HEALTH CARE

- Primary—often outpatient
- Secondary to other diseases (such as inflammation of the front part of the eye, including the iris [anterior uveitis] and corneal ulceration, with or without inflammation [ulcerative keratitis])—may need hospitalization while the underlying problem is diagnosed and treated

ACTIVITY

- Primary—no restriction for most patients
- Suspected contact irritant or sudden (acute) allergic disease—prevent (if possible) contact with the agent causing the irritation or allergy
- Suspected feline herpesvirus (FHV)—minimizing stress is recommended
- Do not expose patients to other cats to decrease risk of spread of infectious causes of inflammation of the moist tissues of the eye (conjunctivitis)

DIET

- Suspected underlying skin disease and/or food allergy—food elimination diet recommended; an “elimination diet” is a diet that does not contain substances that the animal normally eats and is free of additives

SURGERY

- Blockage of the outflow portion of the drainage system that normally moves tears to the nasal passages (known as the “nasolacrimal system”), such as an obstructed nasolacrimal duct—surgical repair is difficult; treatment often not recommended
- Cancer involving the moist tissues of the eye (“conjunctival cancer”)—may involve surgical removal of the tumor followed by radiation therapy; freezing (known as “cryotherapy”); or heating of the tissues using radiofrequency waves (known as “radiofrequency hyperthermia”); may involve surgical removal of the eyeball and associated tissues (known as “enucleation”), depending on the type of tumor and the extent of involvement
- Scar tissue between the eyelid and the eyeball (known as “symblepharon”)—common complication of newborn conjunctivitis; may require surgical removal of the scar tissue, once infection is controlled
- Condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up (known as “corneal edema;” condition known as “corneal sequestration”)—surgical removal of the surface of the cornea (known as “keratectomy”) may be required



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Infection Caused by Feline Herpesvirus (FHV)

- Condition usually mild and may resolve on its own
- Antiviral treatment—indicated for certain situations (such as corneal ulcers caused by FHV and conjunctivitis that does not respond to symptomatic treatment); treatment may be directed at controlling secondary bacterial infection only
- Antiviral medications include 0.1% idoxuridine solution; vidarabine 3% ointment; trifluridine
- Lysine

Bacterial Infections (Chlamydial or Mycoplasmal Infections)

- Antibiotics may be applied directly to the moist tissues of the eye (“topical treatment”) or may be given by mouth (“systemic treatment”)
- Tetracycline eye ointment (Terramycin®)—applied to the moist tissues of the eye (that is, topical treatment); continue for several days past resolution of all clinical signs; recurrence or reinfection common; use of tetracycline administered by mouth (systemic treatment) may be used for difficult cases
- Topical chloramphenicol or ciprofloxacin eye solution is an alternative to topical oxytetracycline eye ointment
- Azithromycin, an antibiotic, may be given by mouth (systemic treatment)

Other Bacterial Infections

- Antibiotics based on bacterial culture and sensitivity results

Newborn Conjunctivitis

- The veterinarian will open the lid margins carefully, establish drainage of discharge, and treat with a topical antibiotic and an antiviral

for suspected feline herpesvirus (FVH)

Eosinophilic Conjunctivitis

- Steroids applied directly to the moist tissues of the eye (“topical treatment”)—usual treatment; 0.1% dexamethasone
- Oral megestrol acetate—may help condition that does not respond to steroids; discuss possible side effects with your pet’s veterinarian
- Other steroids—1% prednisolone acetate; betamethasone; hydrocortisone



FOLLOW-UP CARE

PATIENT MONITORING

- Recheck shortly after beginning treatment (at 5 to 7 days); then recheck as needed

PREVENTIONS AND AVOIDANCE

- Treat any underlying disease that may make the eye disease worse—allergic or immune-mediated skin disease; “dry eye” (KCS)
- Prevent re-exposure to source of infection
- Minimize stress for patients with inflammation of the moist tissues of the eye caused by feline herpesvirus (known as “herpetic conjunctivitis”)
- Isolate patients with infectious conjunctivitis to prevent spread of disease
- Vaccination against viral causes (feline herpesvirus [FHV], calicivirus)—recommended; infection is still possible if the cat was exposed to an infectious agent before being vaccinated (for example, FHV infection from an infected mother cat [queen])

POSSIBLE COMPLICATIONS

- Corneal sequestration (condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up [corneal edema])
- Scar tissue between the eyelid and the eyeball (symblepharon)—common complication of newborn conjunctivitis
- “Dry eye” (KCS)

EXPECTED COURSE AND PROGNOSIS

- Feline herpesvirus (FVH) infection—most patients become long-term (chronic) carriers of the virus; episodes less common as patient matures; may see repeated flare-ups; tend to note more severe clinical signs at times of stress or decreased ability to produce immune response (known as being “immunocompromised”)
- Bacterial infection/inflammation of the moist tissues of the eye (conjunctivitis)—usually resolves with appropriate administration of antibiotics; if an underlying disease is found (such as “dry eye” [KCS]), resolution may depend on appropriate treatment and resolution of the disease
- Immune-mediated diseases (such as eosinophilic conjunctivitis)—diseases tend to be controlled, not cured; may require long-term (chronic) treatment with steroids at the lowest dose possible



KEY POINTS

- If a large amount of discharge is noted, gently clean the eyes before administering treatment
- If both eye solutions and eye ointments are prescribed, apply the solution(s) before applying the ointment(s)
- If several eye solutions are prescribed, wait several minutes between treatments
- Call for instructions if the condition worsens, which indicates that the condition may not be responsive to treatment or may be progressing or that the animal may be having an adverse reaction to a prescribed medication

INFLAMMATION OF THE MOIST TISSUES OF THE EYE (CONJUNCTIVITIS) IN DOGS



BASICS

OVERVIEW

- Inflammation of the moist tissues of the eye (known as the “conjunctiva”); the conjunctiva is the vascularized moist tissue (mucous membrane) that covers the front part of the eyeball or globe, up to the edge of the cornea (known as the “bulbar conjunctiva”) and lines the lids and third eyelid (known as the “palpebral conjunctiva”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predislection

- Breeds susceptible to allergic or immune-mediated skin diseases (such as “atopy”) tend to have more problems with allergic inflammation of the moist tissues of the eye (conjunctivitis) or “dry eye” (known as “keratoconjunctivitis sicca” or “KCS”); “atopy” is a disease in which the animal is sensitized (or “allergic”) to substances found in the environment (such as pollen) that normally would not cause any health problems

SIGNS/OBSERVED CHANGES in the ANIMAL

- Squinting or spasmodic blinking (known as “blepharospasm”)
- Redness of the moist tissues of the eye (known as “conjunctival hyperemia”)
- Discharge from the eye(s); may be clear or may contain mucus and/or pus
- Fluid build-up (known as “edema”) of the moist tissue covering of the eyeball (bulbar conjunctiva), around the cornea (condition known as “chemosis”)
- Follicle formation; the “follicles” are accumulations of lymphoid tissue located at the moist tissue surface of the third eyelid and the eyelids, causing a “cobblestone” appearance; lymphoid tissue contains lymphocytes, a type of white-blood cell that are involved in allergies and response to irritants

CAUSES

Bacterial Causes

- Primary condition (that is, not secondary to another condition such as “dry eye” [keratoconjunctivitis sicca or KCS])—rare
- Newborn inflammation of the moist tissues of the eye (conjunctivitis)—accumulation of discharge, often associated with a bacterial or viral infection; seen before the eyelids separate or open

Viral Causes

- Canine distemper virus

Immune-mediated

- Allergic conjunctivitis—especially in atopic patients; “atopy” is a disease in which the animal is sensitized (or “allergic”) to substances found in the environment (such as pollen) that normally would not cause any health problems
- Follicular conjunctivitis—inflammation of the moist tissues of the eye (conjunctivitis) characterized by accumulations of lymphoid tissue located at the moist tissue surface of the third eyelid and the eyelids, causing a “cobblestone” appearance; lymphoid tissue contains lymphocytes, a type of white-blood cell that are involved in allergies and response to irritants
- Plasma-cell conjunctivitis—inflammation of the moist tissues of the eye (conjunctivitis) characterized by the presence of plasma cells (a specialized type of white-blood cell; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease); especially in German shepherd dogs
- Related to generalized (systemic) immune-mediated diseases—such as pemphigus, in which the body attacks its own tissues **Cancer**
- Tumors involving the moist tissues of the eye (conjunctiva)—rare; include melanoma, hemangioma, hemangiosarcoma, lymphoma, papilloma, and mast-cell tumors
- Lesions that appear to be cancer, but are not cancerous (known as “pseudocancer”)—inflammation of the border between the cornea (the clear part of the eye, located in the front of the eyeball) and the sclera (the white part of the eye, it is composed of a tough covering that protects the eyeball) characterized by the presence of nodules (condition is known as “nodular episcleritis” [also called “fibrous histiocytoma,” “ocular nodular granuloma,” and “conjunctival pseudotumor”]; most commonly seen in collies and mixed collies; believed to be immune-mediated; usually appears as a pink mass

Secondary to Disease of the Tissues Surrounding the Eye (known as “adnexa,” such as eyelids, third eyelid, and tear glands)

- Lack of normal tear film (known as “aqueous tear film deficiency”); “dry eye” (KCS)
- Lid diseases (such as “entropion,” in which the eyelid curls inward, allowing facial hair to rub the eye; “ectropion,” in which the eyelid is turned outward) and lash diseases (such as “distichiasis,” in which two rows of eyelashes are present on a single eyelid; “ectopic cilia,” in which one or more eyelashes grows in an unusual location [may grow through the conjunctiva, leading to irritation of the eye])—may lead to clinical signs of inflammation of the moist tissues of the eye (conjunctivitis)
- Secondary to blockage of the outflow portion of the drainage system that normally moves tears to the nasal passages (known as the “

nasolacrimal system”), such as an obstructed nasolacrimal duct or lack of normal openings on the eyelids into the tear drainage system (known as “imperforate puncta”)

Secondary to Trauma or Environmental Causes

- Foreign body located in the moist tissues of the eye
- Irritation from dust, chemicals, or eye medications

Secondary to Other Eye Diseases

- Disorder of the cornea (the clear outer layer of the front of the eye) characterized by the presence of ulcers, with or without inflammation (condition known as “ulcerative keratitis”)
- Inflammation of the front part of the eye, including the iris (known as “anterior uveitis”)
- Disease of the eye, in which the pressure within the eye is increased (known as “glaucoma”)

RISK FACTORS

- Exposure to dogs with canine distemper viral infections



TREATMENT

HEALTH CARE

- Primary—often outpatient
- Secondary to other diseases (such as inflammation of the front part of the eye, including the iris [anterior uveitis] and corneal ulceration, with or without inflammation [ulcerative keratitis])—may need hospitalization while the underlying problem is diagnosed and treated

ACTIVITY

- Primary—usually no restriction
- Suspected contact irritant or sudden (acute) allergic disease—prevent (if possible) contact with the agent causing the irritation or allergy
- Do not expose patients to other dogs to decrease risk of spread of infectious causes (such as canine distemper virus) of inflammation of the moist tissues of the eye (conjunctivitis)

DIET

- Suspected underlying skin disease and/or food allergy—food elimination diet recommended; “elimination diet” is a diet that does not contain substances that the animal normally eats and is free of additives

SURGERY

- Blockage of the outflow portion of the drainage system that normally moves tears to the nasal passages (known as the “nasolacrimal system”), such as an obstructed nasolacrimal duct—surgical repair is difficult; treatment often not recommended
- Cancer involving the moist tissues of the eye (“conjunctival cancer”)—may involve surgical removal of the tumor followed by radiation therapy; freezing (known as “cryotherapy”); or heating of the tissues using radiofrequency waves (known as “radiofrequency hyperthermia”); may involve surgical removal of the eyeball and associated tissues (known as “enucleation”), depending on the type of tumor and the extent of involvement



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Bacterial Infections

- Antibiotics based on bacterial culture and sensitivity results
- Antibiotics may be applied directly to the moist tissues of the eye (“topical treatment”) or may be given by mouth (“systemic treatment”)
- Initial treatment—broad-spectrum topical antibiotic or specific antibiotic based on results of microscopic examination of discharge and/or conjunctival scraping, while waiting for bacterial culture and sensitivity results; may try treatment based on experience with other cases of conjunctivitis, performing a bacterial culture and sensitivity only if patient does not respond to selected treatment
- Topical triple antibiotic or chloramphenicol—if cocci (general type of bacteria) seen on microscopic examination of discharge and/or conjunctival scraping
- Gentamicin or tobramycin—if rods (general type of bacteria) seen on microscopic examination of discharge and/or conjunctival scraping
- Ciprofloxacin—may be useful for severe bacterial inflammation of the moist tissues of the eye (conjunctivitis)
- Systemic antibiotics—occasionally indicated, especially for more generalized disease (such as inflammation of the moist tissues of the eye [conjunctivitis] associated with skin infection characterized by the presence of pus [known as “pyoderma”])

Newborn Conjunctivitis

- The veterinarian will open the lid margins carefully, establish drainage of discharge, and treat with topical antibiotic

Immune-mediated Conjunctivitis

- Depends on severity
- Topical steroids—applied directly to the moist tissues of the eye; 0.1% dexamethasone; improve clinical signs of allergic, follicular conjunctivitis (characterized by accumulations of lymphoid tissue located at the moist tissue surface of the third eyelid and the eyelids, causing a “cobblestone” appearance), and plasma-cell conjunctivitis (characterized by the presence of plasma cells); improvement often temporary
- Treatment of any underlying disease (such as atopy) often improves clinical signs; “atopy” is a disease in which the animal is sensitized (or “allergic”) to substances found in the environment (such as pollen) that normally would not cause any health problems
- Other steroids—1% prednisolone acetate; betamethasone; hydrocortisone



FOLLOW-UP CARE

PATIENT MONITORING

- Recheck shortly after beginning treatment (at 5 to 7 days); then recheck as needed

PREVENTIONS AND AVOIDANCE

- Treat any underlying disease that may make the eye disease worse—allergic or immune-mediated skin disease; “dry eye” (KCS)
- Vaccination against canine distemper virus

EXPECTED COURSE AND PROGNOSIS

- Bacterial infection/inflammation of the moist tissues of the eye (conjunctivitis)—usually resolves with appropriate administration of antibiotics; if an underlying disease is found (such as “dry eye” [KCS]), resolution may depend on appropriate treatment and resolution of the disease
- Immune-mediated diseases—diseases tend to be controlled, not cured; may require long-term (chronic) treatment with steroids at the lowest dose possible



KEY POINTS

- If a large amount of discharge is noted, gently clean the eyes before administering treatment
- If both eye solutions and eye ointments are prescribed, apply the solution(s) before applying the ointment(s)
- If several eye solutions are prescribed, wait several minutes between treatments
- Call for instructions if the condition worsens, which indicates that the condition may not be responsive to treatment or may be progressing or that the animal may be having an adverse reaction to a prescribed medication
- An Elizabethan collar should be placed on the patient, if self-trauma occurs

CONSTIPATION AND OBSTIPATION



BASICS

OVERVIEW

- “Constipation” is infrequent, incomplete, or difficult defecation with passage of hard or dry bowel movement (feces)
- “Obstipation” is constipation that is difficult to manage or does not respond to medical treatment, caused by prolonged retention of hard, dry bowel movement (feces); defecation is impossible in the patient with obstipation

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- More common in cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Straining to defecate with small or no fecal volume
- Hard, dry bowel movement (feces)
- Infrequent defecation
- Small amount of liquid stool with mucus in it—sometimes with blood present, produced after prolonged straining to defecate (known as “tenesmus”)
- Occasional vomiting, lack of appetite, and/or depression
- Large bowel (colon) filled with hard bowel movement (feces)
- Other signs depend on cause
- Rectal examination may reveal a mass in the rectum or large intestine (colon); narrowing of the colon (known as a “colonic stricture”); perineal hernia, which develops when the muscles supporting the rectum weaken and separate, allowing the rectum and/or bladder to slide under the skin and causing swelling in the area of the anus; anal sac disease; presence of a foreign body or material; enlarged prostate; or narrowed pelvic canal

CAUSES

Dietary Causes

- Bones
- Hair
- Foreign material
- Excessive fiber
- Inadequate water intake

Environmental Causes

- Lack of exercise
- Change of environment—hospitalization, dirty litter box
- Inability to walk

Drugs

- Anticholinergics (medications used as preanesthetics or to treat diarrhea, such as atropine)
- Antihistamines
- Opioids
- Barium sulfate
- Sucralfate (medication that forms a protective barrier over gastrointestinal ulcers)
- Antacids
- Kaopectolin
- Iron supplements
- Medications to remove excess fluid from the body (known as “diuretics”)

Painful Defecation

- Disease of the anus and/or rectum—inflammation of the anal sacs (known as “anal sacculitis”); anal-sac abscess; one or multiple draining tracts around the anus (known as “perianal fistulae”); narrowing of the anus (known as an “anal stricture”); anal spasm; rectal foreign body; condition in which the rectum slips out of its normal position and protrudes through the opening of the anus (known as a “rectal prolapse”); condition in which bowel movement (feces) becomes trapped and matted in the hair around the anus, blocking the anus (known as “pseudocoprostasis”); and inflammation of the lining of the rectum (known as “proctitis”)
- Trauma—fractured pelvis; fractured limb; dislocated hip; bite wound or laceration in the tissue around the anus; perineal (area between the anus and external genitalia) abscess

Mechanical Obstruction

- Extraluminal (outside of the “tube” of the intestinal tract)—healed pelvic fracture with narrowed pelvic canal; enlarged prostate; inflammation of the prostate (known as “prostatitis”); prostate cancer; condition in which bowel movement (feces) becomes trapped and matted in the hair around the anus, blocking the anus (pseudocoprostasis); enlarged lymph nodes
- Intraluminal (inside the “tube” of the intestinal tract) and intramural (within the wall of the intestines)—colonic or rectal cancer or

polyp; narrowing of the rectum (known as a “rectal stricture”); foreign body in the rectum; presence of a pouch or sac-like opening from the rectum (known as a “rectal diverticulum”); condition in which muscles supporting the rectum weaken and separate, allowing the rectum and/or bladder to slide under the skin and causing swelling in the area of the anus (known as a “perineal hernia”); condition in which the rectum slips out of its normal position and protrudes through the opening of the anus (rectal prolapse); and congenital (present at birth) defect in which the anus does not have an opening (known as “atresia ani”)

Disease of the Nerves and/or Muscles

- Central nervous system—paralysis of the rear legs (known as “paraplegia”); spinal cord disease; intervertebral disk disease; brain disease (such as lead toxicity or rabies)
- Peripheral nervous system—abnormal function of the autonomic nervous system (known as “dysautonomia”); sacral nerve disease; sacral nerve trauma (such as tail fracture/pull injury)
- Smooth-muscle dysfunction of the large bowel (colon)—enlarged large intestine of unknown cause (so called “idiopathic megacolon”) in cats

Metabolic and Hormonal Disease

- Impaired smooth-muscle function of the large bowel (colon)—low levels of thyroid hormone (known as “hypothyroidism”); low levels of potassium in the blood (known as “hypokalemia”) as in long-term (chronic) kidney failure; high levels of calcium in the blood (known as “hypercalcemia”); high levels of parathyroid hormone in the blood (known as “hyperparathyroidism”)—parathyroid hormone regulates calcium levels in the blood by causing calcium to be reabsorbed from bone
- Debility—general muscle weakness, dehydration, cancer

RISK FACTORS

- Drug therapy—anticholinergics (medications used as preanesthetics or to treat diarrhea, such as atropine), narcotics, barium sulfate
- Diseases causing dehydration
- Intact male—perineal hernia (condition in which muscles supporting the rectum weaken and separate, allowing the rectum and/or bladder to slide under the skin and causing swelling in the area of the anus), prostate disease
- Perianal fistula (one or multiple draining tracts around the anus)
- Eating of nonfood items (known as “pica”)—foreign material
- Excessive grooming—hair ingestion
- Decreased grooming/inability to groom—long-haired cats
- Pelvic fracture



TREATMENT

HEALTH CARE

- Remove or treat any underlying cause, if possible
- Discontinue any medications that may cause constipation
- May need to treat as inpatient if pet has obstipation (constipation that is difficult to manage or does not respond to medical treatment) and/or dehydration
- Fluids for dehydrated patients

ACTIVITY

- Encourage activity

DIET

- Dietary supplementation with a bulk-forming agent (such as bran, methylcellulose, canned pumpkin, psyllium) often is helpful, though these agents can sometimes worsen fecal distension within the colon; if this occurs, feed a low residue-producing diet

SURGERY

- Manual removal of feces with the animal under general anesthesia (after rehydration) may be required, if enemas and medications are unsuccessful
- Surgical procedure to remove part of the colon (known as a “subtotal colectomy”) may be required with recurring obstipation (constipation that is difficult to manage or does not respond to medical treatment)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Emollient laxatives—docusate sodium or docusate calcium
- Stimulant laxatives—bisacodyl
- Saline laxatives

- Disaccharide laxative—lactulose
- Warm-water enemas may be needed
- Suppositories can be used as a replacement for enemas, such as glycerol, bisacodyl, or docusate sodium products
- Intestinal motility modifiers can be tried—cisapride may stimulate motility; indicated with early enlargement of the large intestine or colon (megacolon)



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor frequency of defecation and stool consistency at least twice a week initially, then weekly or biweekly

PREVENTIONS AND AVOIDANCE

- Feed appropriate diet and keep pet active

POSSIBLE COMPLICATIONS

- Long-term (chronic) constipation or recurrent obstipation (constipation that is difficult to manage or does not respond to medical treatment) can lead to acquired (condition that develops sometime later in life/after birth) enlargement of the large intestine or colon (megacolon)
- Overuse of laxatives and enemas can cause diarrhea
- Lining of the large bowel (colon) can be damaged by improper enema technique, repeated rough mechanical breakdown of bowel movement (feces), or pressure of hard feces
- Inability to control bowel movements (known as “fecal incontinence”)

EXPECTED COURSE AND PROGNOSIS

- Vary with underlying cause



KEY POINTS

- Feed appropriate diet and encourage activity

COONHOUND PARALYSIS (IDIOPATHIC POLYRADICULONEURITIS)



BASICS

OVERVIEW

- Sudden (acute) inflammation of multiple nerve roots and peripheral nerves in dogs, with or without a previous history of contact with a raccoon
- Coonhound paralysis generally refers to dogs that have had previous history of contact with a raccoon, while sudden (acute) canine idiopathic polyradiculoneuritis refers to dogs that have the same nervous system signs and progression of disease, but do not have history or contact with a raccoon
- “Idiopathic” is the medical term for a disease of unknown cause; “polyradiculoneuritis” is the medical term for inflammation of multiple spinal nerve roots and nerves
- Proposed animal model for Guillain-Barré syndrome in people

GENETICS

- No proven basis

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Coonhound paralysis—coonhounds; any breed in contact with raccoons are susceptible
- Sudden (acute) canine idiopathic polyradiculoneuritis—no breed predilection

SIGNS/OBSERVED CHANGES in the ANIMAL

- Appear 7 to 14 days after contact with a raccoon (coonhound paralysis)
- Stiff-stilted gait in all four limbs—initially
- Rapid progression to a flaccid lower motor neuron weakness to partial paralysis of all four legs (known as “tetraparesis”) to paralysis of all four legs (known as “tetraplegia”); “lower motor neuron disease” involves the nerves that connect the spinal cord and muscles
- Appetite and water consumption—usually normal
- Urination and defecation—normal
- Initial progression—usually occurs over 4 to 5 days; maximum progression can take up to 10 days
- Usually symmetrical nervous system signs
- Generalized decreased reflexes (known as “hyporeflexia”) to absence of reflexes (known as “areflexia”); decreased muscle tone (known as “hypotonia”) to lack of muscle tone (known as “atonia”), and severe decrease in muscle mass due to lack of nerve stimulation (known as “severe neurogenic muscle atrophy”)
- Rear legs more severely affected than are the front legs in a few patients
- Breathing—labored in severely affected dogs; occasional progression to paralysis of the respiratory muscles
- Loss of voice (known as “aphonia”) or altered voice (known as “dysphonia”) is common
- Weakness of facial muscles (known as “facial paresis”)—dog may not be able to completely close the eyelids
- Pain sensation is intact (in other words, dog can still feel pain); commonly dog may be overly sensitive to pain or touch (known as “hyperesthesia”)
- Motor dysfunction—always predominates; even a dog that is paralyzed in all four legs (tetraplegia) can usually wag its tail
- Sudden (acute) canine idiopathic polyradiculoneuritis—nervous system signs and progression of disease is the same, except no known initial encounter with a raccoon is identified

CAUSES

- Coonhound paralysis—contact with a raccoon; perhaps more important, contact with raccoon saliva
- Sudden (acute) canine idiopathic polyradiculoneuritis—none proven; possibly previous respiratory or gastrointestinal viral or bacterial infection, or vaccination

RISK FACTORS

- Coonhound paralysis—coonhounds tend to be susceptible, primarily because of the nature of their activities; previous disease does not confer immunity and may increase risk of redevelopment; multiple bouts are not uncommon
- Sudden (acute) canine idiopathic polyradiculoneuritis—unknown



TREATMENT

HEALTH CARE

- Inpatient—closely monitor patients in the progressive stage of the disease (especially during the first 4 days) for breathing problems
- Severe breathing compromise—intensive care; breathing support and oxygen, as required
- Intravenous fluid therapy—lactated Ringer’s solution; necessary only if patient is dehydrated because of inability to reach water
- Outpatient—stabilize patient, after initial diagnostic confirmation of disease
- Dogs usually are able to eat and drink, if they can reach the food and water; often must be hand fed because of paralysis
- Intensive physical therapy—important to decrease loss of muscle mass (muscle atrophy)
- Frequent turning of the dog and excellent padding of the bed—essential to prevent pressure sores

ACTIVITY

- Encourage as much movement as possible; many patients are paralyzed in all four legs (tetraplegia)

DIET

- No restrictions
- Make sure dog is able to reach food and water
- If the dog has weakness involving the neck muscles—may need to be hand fed



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- None proven effective
- Immunoglobulin—given early may decrease severity and/or shorten recovery time; “immunoglobulin” is an immune protein or antibody necessary for fighting disease



FOLLOW-UP CARE

PATIENT MONITORING

- Outpatient—keep in close contact with veterinarian regarding complications or changes in the dog’s condition
- Urinalysis—perform periodically to check for inflammation of the bladder (known as “cystitis”) in dogs that are paralyzed in all four legs (tetraplegia) or severely weak or partially paralyzed in all four legs (tetraparesis)
- Ideally, re-evaluate at least every 2 to 3 weeks

PREVENTIONS AND AVOIDANCE

- Coonhound paralysis—avoid contact with raccoons; often not feasible because of coonhounds’ environment and primary use as raccoon hunters
- Sudden (acute) canine idiopathic polyradiculoneuritis—none

POSSIBLE COMPLICATIONS

- Paralysis of respiratory muscles—in progressive stage of the disease
- Pressure sores; skin lesions that develop due to contact with urine, when the hair and skin remain damp (known as “urine scald”); and inflammation of the bladder (cystitis)—common in dogs that are recumbent for a prolonged time

EXPECTED COURSE AND PROGNOSIS

- Most recover fully
- Mild residual nervous system deficits—duration of several weeks in mildly to moderately affected dogs; duration of 3 to 4 months with severe disease



KEY POINTS

- Good nursing care is essential
- Prevent pressure sores and skin lesions that develop due to contact with urine, when the hair and skin remain damp (urine scald)
- Limit the degree of loss of muscle mass (muscle atrophy) by diligent physical therapy (such as passive limb movement and swimming as the patient's strength begins to improve)
- The dog needs soft (fleeces are excellent), resilient bedding (straw is excellent) that must be kept clean and free of urine and feces, frequent turning (every 3 to 4 hours), frequent bathing, and adequate nutrition

COPPER-STORAGE LIVER DISEASE



BASICS

OVERVIEW

- Abnormal accumulation of copper in the liver, causing sudden (acute) inflammation of the liver (hepatitis) or long-term (chronic) hepatitis and eventually progressive damage and scarring of the liver (known as “cirrhosis”)
- Primary disease is thought to be the result of genetic-based abnormal copper metabolism
- Most of the following information is based on studies from affected Bedlington terriers

GENETICS

- Autosomal recessive trait in Bedlington terriers due to the lack of a specific gene (COMMD1) coding for a protein involved in excretion of copper in the bile, which is produced by the liver
- The mode of inheritance in West Highland white terrier, Skye terrier and other breeds affected is unknown
- Dalmatians, Doberman pinschers and Labrador retrievers also have breed-related chronic hepatitis with copper accumulation (suspected to be a genetic disorder)
- Isolated dogs of other breeds with liver disease have been found to have elevated liver copper concentrations, but little evidence supports a genetic basis in these dogs
- Bedlington terrier—at one time, possibly as many as two-thirds of Bedlington terriers either were carriers of the gene or were affected by the disease; with recent genetic screening, the incidence is now much lower
- The prevalence in certain lines of West Highland white terrier appears to be high, but the incidence in all West Highland white terriers is low
- Reported 4% to 6% of Doberman pinschers may have chronic hepatitis
- The incidence in other breeds is unknown

SIGNALMENT/DESCRIPTION of ANIMAL

- Dogs
- Rare, single isolated cases have been reported of abnormal copper accumulation in the liver of cats

Breed Predislection

- Bedlington terriers, West Highland white terriers, Skye terriers, Doberman pinschers, Dalmatians and Labrador retrievers are reported to have increased liver copper concentrations

Mean Age and Range

- Bedlington terrier—copper accumulates over time to a maximum level at about 6 years of age
- Dogs can be affected clinically at any age, though most present as middle-aged to older dogs having chronic hepatitis
- West Highland white terrier—maximum copper accumulation may be observed by 12 months of age, but clinical disease can occur at any time
- Skye terrier—all ages can be affected
- Doberman pinschers are reported to begin to develop hepatitis and copper accumulation at 1 to 3 years of age
- Labrador retrievers and Dalmatians are generally middle-aged when diagnosed with clinical disease

Predominant Sex

- Doberman pinscher—females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Primary copper liver diseases (liver diseases are known as “hepatopathies”) generally fall in one of three categories: 1. subclinical disease (condition where the disease is present in the organ or body, but not detectable by abnormal signs or changes in the animal), 2. sudden (acute) disease (an uncommon finding) in which signs are observed most frequently in young dogs associated with acute death of liver tissue (known as “hepatic necrosis”), or 3. long-term (chronic) progressive disease in which signs are observed in middle-aged and older dogs with chronic inflammation of the liver (hepatitis) and damage and scarring of the liver (cirrhosis)
- Secondary copper liver diseases (hepatopathies) present with chronic progressive signs of liver disease due to chronic inflammation of the liver (hepatitis) or progressive damage and scarring of the liver (cirrhosis)
- Acute signs—sudden onset of sluggishness (lethargy), lack of appetite (anorexia), depression, and vomiting; weakness, and yellowish discoloration to skin and moist tissues (icterus or jaundice); pale moist tissues of the body (mucous membranes) due to low red blood cell count (anemia) and dark urine (due to the presence of bilirubin in the urine [bilirubinuria] and hemoglobin in the urine [hemoglobinuria]) in some dogs; many of these dogs have a rapid course and die despite intensive supportive treatment
- Chronic signs—history of waxing and waning sluggishness (lethargy), depression, lack of appetite (anorexia), and weight loss; vomiting, diarrhea, and excessive thirst (polydipsia) and excessive urination (polyuria) may be seen; later signs may include abdominal distention due to fluid build up in the abdomen (ascites), yellowish discoloration to skin and moist tissues (icterus or jaundice), spontaneous bleeding, black or tarry stools (melena), and nervous system signs due to the liver being unable to breakdown ammonia in the body (known as “hepatic encephalopathy”)

CAUSES

- Primary—unknown in all but the Bedlington terrier; copper-storage liver disease in other breeds is suspected to be the result of abnormal liver copper metabolism or excretion defect
- Secondary—liver disease in which the flow of bile is slowed or stopped is known as “cholestatic liver disease;” the abnormal flow of bile results in secondary copper retention

RISK FACTORS

- Primary—feeding high-copper diets, or stress factors that may precipitate acute disease



TREATMENT

HEALTH CARE

- Most dogs are treated as outpatients
- Inpatient evaluation and treatment are needed for dogs with signs of liver failure
- Treatment is determined by the type of disease: acute or chronic hepatitis or liver scarring/cirrhosis
- Animals in liver failure will require fluids and electrolytes

ACTIVITY

- Normal

DIET

- Low-copper diets should be fed to affected animals; however, almost all commercially available diets contain an excess of copper
- Balanced homemade diets avoiding copper-rich foods (such as organ meats) may be used
- Avoid mineral supplements containing copper
- Frequently, feeding a low-copper diet is not feasible, and commercial diets must be used
- Use of specific chemicals to tie up the copper in the system and to allow it to be removed from the body (known as “chelation therapy”) in conjunction with commercial diets has been successful in management of affected Bedlington terriers
- A high-quality, protein-sufficient, moderate-fat-containing diet should be fed to meet caloric needs; protein content should be reduced only when the patient exhibits protein intolerance (that is, has signs of liver-related central nervous system disease [hepatic encephalopathy])
- Water-soluble vitamins should be supplemented under the direction of your pet’s veterinarian

SURGERY

- Liver biopsy may be needed to screen dogs for copper-storage liver disease and to monitor response to treatment
- Animals with liver failure are surgical and anesthetic risks



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- d-Penicillamine chelates copper (that is, ties up copper and allows it to be removed from the body) and promotes excretion of copper into the urine and is suspected to have other copper-protective effects; treatment should be initiated in affected dogs having abnormal hepatic copper concentrations
- Trientine hydrochloride is an alternative copper chelator that appears to be as effective as d-penicillamine
- Zinc reduces intestinal absorption of copper; may be beneficial in affected dogs in the early stages of copper-storage disease
- Use of chelators and zinc at the same time is not recommended and may decrease effectiveness of either drug
- d-alpha tocopherol (vitamin E) may protect the liver from damage caused by copper and is suggested as an additional therapy; vitamin E supplementation should be under the direction of your pet’s veterinarian
- Other antioxidants, such as s-adenosylmethionine (SAME) or silibin (milk thistle), may be beneficial; ask your pet’s veterinarian for recommendations



FOLLOW-UP CARE

PATIENT MONITORING

- Blood tests to monitor levels of liver enzymes every 4 to 6 months

- Monitor body weight
- Measure liver copper concentration within 1 year and thereafter as required by clinical findings
- When using zinc therapy, assess serum zinc concentration every 2 to 3 weeks, until concentration is stable and in desired range and then every 4 to 6 months
- Following therapy (6 months to 1 year) dog should be re-biopsied to monitor therapy; chelation therapy in affected dogs (Bedlington terriers, Doberman pinschers and Labrador retrievers) results in improvement of the hepatitis as seen on biopsy sections using a microscope

PREVENTIONS AND AVOIDANCE

- Breed only Bedlington terriers that do not carry the gene causing the disease; a liver registry is available for Bedlington terriers that are proven unaffected on the basis of liver copper concentration less than 400 μ g/g DW at 1 year of age or DNA gene evaluation

POSSIBLE COMPLICATIONS

- d-Penicillamine can cause lack of appetite (anorexia) and vomiting; d-penicillamine may, in rare cases, cause an autoimmune-like blistering (vesicular) disease of the areas where the skin meets the moist tissues of the body, such as the lips, (known as “mucocutaneous junctions”) that resolves on withdrawal of the drug
- Excess zinc concentrations can cause a breakdown of red blood cells (hemolytic anemia)

EXPECTED COURSE AND PROGNOSIS

- The prognosis is poor in acutely affected young dogs with severe liver failure or older dogs with progressive damage and scarring of the liver (cirrhosis)
- Young dogs with mild-to-moderate acute liver failure usually respond to chelation therapy; the prognosis is fair for these animals; the prognosis is good if the disease is detected before inflammatory changes are noted in the liver, and the dog is started on appropriate therapy



KEY POINTS

- All Bedlington terriers should be screened by using either DNA genetic markers or liver biopsy
- Other breeds should be monitored for abnormal liver enzymes or by liver biopsy
- Therapy is needed for life
- Affected animals should not be used for breeding

PICA AND COPROPHAGIA (Eating Nonfood Items and Eating Feces)



BASICS

OVERVIEW

- Eating of nonfood items (known as “pica”), including eating of feces or bowel movement (known as “coprophagia”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Oriental breeds of cat tend to eat fabrics
- Nursing dogs frequently eat the feces of their puppies; females, whether intact or spayed, are more likely to exhibit coprophagia

SIGNS/OBSERVED CHANGES in the ANIMAL

- Eating of nonfood items (for example, dogs—rocks and feces; cats—fabrics and plastics)
- Bad breath (halitosis), if problem is coprophagia
- Pale moist tissues of the body (mucous membranes) and weakness if the animal has a low red-blood cell count (anemia)
- Thin body condition if signs are accompanied by abnormal digestion or absorption of food (maldigestion or malabsorption)
- Nervous system signs if behavior caused by neurologic disease

CAUSES

Behavioral Causes

- Nest cleaning
- Displacement activity from unavailable herbivore feces
- Responding to punishment, by removing evidence of soiling
- Imitating owners’ behavior—cleaning the nest
- Compulsive behavior
- Attention-seeking behavior

Medical Causes

- Low levels of digestive enzymes produced by the pancreas (known as “exocrine pancreatic insufficiency”)
- Inflammatory bowel disease
- Excessive number of bacteria in the small intestine (known as “small intestinal bacterial overgrowth”)
- Enlarged esophagus (known as “megaesophagus”) and/or narrowing of a section of the esophagus (known as “esophageal stricture”)
- Intestinal parasitism
- Excessive production of thyroid hormone (known as “hyperthyroidism”)
- Diabetes mellitus
- Excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Dietary deficiencies—unproven
- Drug induced (such as effect of steroids, progestins, phenobarbital)
- Low red-blood cell count (anemia)—iron deficiency, other
- Abnormal digestion or absorption of food (maldigestion or malabsorption; for example, secondary to exocrine pancreatic insufficiency)
- Hunger
- Nervous system disease—primary central nervous system or secondary to abnormal circulation in the liver (portosystemic shunt) in which excessive ammonia builds up in the body, leading to nervous system signs

Drug-induced Causes

- Administration of steroids or progestins frequently leads to increased appetite and excessive eating (polyphagia) in dogs

RISK FACTORS

- Early weaned oriental-breed cats fed low-roughage diets with no access to prey or grass may be at risk for wool eating
- Confinement of dogs in barren yards with no environmental stimulation or enrichment—especially predisposes to coprophagia
- Underlying disease leading to low red-blood cell counts (anemia), abnormal digestion or absorption of food (maldigestion or malabsorption)



TREATMENT

HEALTH CARE

- Varies depending on whether the cause is medical or behavioral
- Treat any underlying disease (such as hormonal problems, gastrointestinal disease, or disorders of the pancreas) and withdraw any drugs that could cause increased appetite (polyphagia)
- Correct any dietary deficiencies
- When no underlying medical cause exists—(1) limit access to nonfood items to prevent animal from eating them; (2) find a safe substitute that the animal can eat; and (3) change the animal's motivation to ingest the nonfood item

TREATMENT of EATING of NONFOOD ITEMS (PICA)

- If pica is attention-getting, muzzle dog to prevent ingestion and ignore attempts to obtain nonfood items
- Fabric chewing in cats can be treated by: (1) removing plastic and woolen clothes from the cat's environment; (2) applying a pungent or bitter taste to objects, which may discourage consumption; (3) feeding a high-roughage diet or tough meat to chew, or providing a garden of grass or catnip to graze on (interactive owner punishment is not recommended and may increase stress and anxiety that underlie compulsive disorders)

TREATMENT of EATING FECES of BOWEL MOVEMENT (COPROPHAGIA)

- Can treat coprophagia in a number of ways—can decrease access to feces by prompt disposal; walk dogs on a leash to facilitate removal from vicinity of feces
- Use a muzzle or head halter on walks; give the dog a food reward when it defecates, thereby counterconditioning it to expect food rather than search for feces; other recommendations, although unsupported by any published data, include feeding a less digestible diet; use of a meat tenderizer or pancreatic enzymes; and sprinkling noxious tasting/smelling substances on feces
- Bitter and hot substances, such as quinine, cayenne pepper, and commercial products (for example, FOR-BID™, Alpar Laboratories, Inc., La Grange, IL) have yielded variable results
- Taste-aversion learning is another potentially effective method; your pet's veterinarian may suggest treating the feces with an agent that makes the pet nauseated for a short period of time and after a few experiences of coprophagy followed by nausea and not feeling well, the dog may learn to avoid eating feces

DIET

- Feed a good quality, balanced diet
- May need high-fiber or roughage diet



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- If the problem is a compulsive disorder, the animal's motivation may be changed by administering psychologically active drugs, such as clomipramine, tricyclic antidepressants (TCAs), or selective serotonin reuptake inhibitors (SSRIs)
- Cyproheptadine



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor and record abnormal eating habits to determine if the animal's pica or coprophagia is decreasing
- Discuss progress in controlling abnormal eating habits with your pet's veterinarian in 10–14 days
- If dietary management changes did not markedly improve the problem, further diagnostic testing and/or medication may be needed

PREVENTIONS AND AVOIDANCE

- Limit access to nonfood items to prevent animal from eating them
- Find a safe substitute that the animal can eat
- Remove plastic and woolen material from the cat's environment
- Apply a pungent or bitter taste to objects, which may discourage consumption

POSSIBLE COMPLICATIONS

- Gastrointestinal complications—foreign bodies, diarrhea, vomiting, bad breath (halitosis)

EXPECTED COURSE AND PROGNOSIS

- Realistic expectations must be understood; changing a behavior that has become a habit is very challenging
- Immediate control of a long-standing problem is unlikely



KEY POINTS

- Ignore the behavior as much as possible and avoid rewarding the behavior
- Abnormal behavior should be evaluated by your veterinarian as soon as possible to determine if a physical cause exists
- Treatment may include behavioral modification and psychologically active drugs if no physical cause is identified
- Realistic expectations must be understood; changing a behavior that has become a habit is very challenging

COUGH



BASICS

OVERVIEW

- A sudden, forceful exhaling action (expiration) of air through the airway opening (glottis) of the voice box, preceded by an exaggerated effort to inhale (inspiration) and usually accompanied by an audible sound

SIGNALMENT/DESCRIPTION of ANIMAL

- Dogs and cats of all ages and breeds

SIGNS/OBSERVED CHANGES in the ANIMAL

- Cough
- Collapse
- Vomiting or retching

CAUSES

Cough is a sign that results from many conditions. These include diseases or abnormalities in the upper airway (respiratory tract) consisting of the nose and throat (known as the “nasopharyngeal” area), the voice box (larynx), and the windpipe (trachea). The lower respiratory tract consists of the lungs and related tissues (such as the bronchi). In addition, problems involving the lungs and the blood vessels in the lungs (pulmonary/vascular disease) can lead to coughing as can problems in the esophagus and lining of the chest.

Upper Respiratory Tract Diseases

- Nasopharyngeal area (nose and throat)—inflammation of the nose (rhinitis); inflammation of the sinuses (sinusitis); foreign body or tumor in the nose or throat; inflammation of the tonsils (tonsillitis); tumor of the tonsils—due to extension of inflammation and/or secretions into the throat and/or voice box
- Larynx (voice box)—inflammation; foreign body; trauma; tumors
- Trachea (windpipe)—inflammation (tracheitis); infections (viral, bacterial, and parasitic); foreign body; collapse of the windpipe (tracheal collapse); tumor

Lower Respiratory Tract Diseases

- Bronchial—inflammation; infection (viral, bacterial, and parasitic); allergy; foreign body; tumors or cancer
- Pulmonary (lung)—inflammation; infection (viral, bacterial, and fungal); aspiration pneumonia; fluid build-up in the lungs (pulmonary edema); cancer (may originate in the lungs [primary cancer] or have spread into the lungs [metastatic cancer])
- Pulmonary/vascular (lung/blood vessels)—heartworm disease; blood clots in the lungs (thrombosis or embolism); congestive heart failure (CHF); high blood pressure in the lungs (pulmonary hypertension); heart tumors

Other Diseases

- Esophagus—inflammation; foreign body; tumor
- Diseases of the lining of the chest (pleural diseases) pressing the airways/lung tissue together (airway compression)—inflammation; infection (bacterial and fungal); hernia; tumor

RISK FACTORS

- Congenital (present at birth) and acquired (develop later in life/after birth) disorders of the esophagus, stomach, and upper gastrointestinal tract may lead to aspiration pneumonia
- Excessive levels of steroids produced by the adrenal glands (disease known as “hyperadrenocorticism” or “Cushing’s disease”) or chronic administration of steroids in treating various disease conditions—may increase incidence of blood clots to the lungs (pulmonary thromboembolism) and may decrease resistance to respiratory infections
- Certain inherited heart disorders—increases risk of fluid build-up in the lungs (pulmonary edema) secondary to congestive heart failure
- Environmental factors—exposure to certain viral, bacterial, fungal, and parasitic diseases; exposure to infected animals; exposure of dogs or cats to mosquitoes (when effective heartworm preventive medication has not been administered at all or routinely to the dog or cat)



TREATMENT

HEALTH CARE

- Outpatient—unless congestive heart failure is diagnosed, oxygen therapy is required, or marked alteration in breathing ability or coughing up/spitting up blood (hemoptysis) is observed
- Wide variety of conditions can be responsible for cough, and a fairly extensive workup may be required to define and treat the underlying cause

ACTIVITY

- Exercise restriction—best enforced until a cause is established and corrected, especially when activity aggravates the condition

DIET

- Normal for many causes of cough
- If cause of cough is congestive heart failure, your pet's veterinarian may recommend low dietary sodium levels

SURGERY

- Surgical intervention—primary indications include collapse of the windpipe that does not respond to medical treatment (known as “refractory tracheal collapse”), paralysis of the voice box (laryngeal paralysis), and tumors involving the respiratory tract



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Symptomatic treatment—drugs to open up (dilate) the bronchi (bronchodilator) and to increase secretions in the bronchi and to aid in the clearing of the lungs (expectorant therapy)
- Broad-spectrum antibiotics—for suspected infection when results of bacterial culture and sensitivity testing are pending
- Bronchodilators (such as extended-release theophylline and terbutaline) with or without the use of expectorants—may be beneficial for a variety of diseases affecting the trachea and lower respiratory airways
- Corticosteroids may be the only effective therapy in the long-term management of chronic bronchitis in dogs and asthma in domestic cats; oral or inhaled corticosteroids can be effective in controlling cough
- Cough suppressants (such as hydrocodone and torbutrol)—avoid in patients with cough secondary to bacterial respiratory infection and congestive heart failure; while cough suppressants may prove beneficial in controlling coughs of other origin, use should be withheld until thorough diagnostic procedures have been completed
- Therapeutic drainage of fluid within the chest cavity (known as “thoracentesis”)—perform on any patient in which build-up of fluids in the space between the lungs and chest wall (the fluid is known as “pleural effusion”) is felt to be contributing to any breathing discomfort or distress



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor control of the cough
- Follow-up chest X-rays in 3 to 7 days with heart and/or blood vessel disease, in 10 to 14 days with lung disease; in 3 to 4 weeks to monitor potential tumors

PREVENTIONS AND AVOIDANCE

- Prevention is determined by the cause of the cough; use of heartworm preventives to protect the dog or cat from heartworm infection; vaccinations and limiting exposure to pets with infectious disease coughing (such as kennel cough for dogs)

POSSIBLE COMPLICATIONS

- Resolution or medical control of cough does not ensure complete elimination of the underlying cause
- Serious respiratory dysfunction and even death may be caused by underlying disease
- Complications of the cough—spread respiratory infections to other dogs or cats, complicate inflammatory conditions in the airways, or even result in emphysema or pneumothorax (air in the chest cavity) by causing rupture of diseased areas of lung tissue

EXPECTED COURSE AND PROGNOSIS

- The expected course and prognosis are determined by the cause of the cough
- Serious respiratory dysfunction and even death may be caused by underlying disease



KEY POINTS

- Cough is a sign of disease
- Wide variety of conditions can be responsible for cough, and a fairly extensive workup may be required to define and treat the

underlying cause

- Treatment is based on diagnosis of underlying cause

CRANIAL CRUCIATE LIGAMENT DISEASE



BASICS

OVERVIEW

- The “stifle” is the knee joint of the dog; it is the joint between the large upper thigh bone (the femur) and the two lower leg bones (tibia and fibula)
- A “ligament” is a band of connective or fibrous tissue that connects two bones or cartilage at a joint; the “cranial cruciate ligament” is the ligament that connects the inner, back part of the femur with the tibia—it helps to stabilize the stifle joint
- “Cranial cruciate ligament disease” is the sudden (acute) or progressive failure of the cranial cruciate ligament, which results in partial to complete instability of the stifle joint
- “Cranial cruciate rupture” is the tearing of the cranial cruciate ligament; it is the most common cause of rear-leg lameness in dogs and a major cause of degenerative joint disease (progressive and permanent deterioration of joint cartilage) in the stifle joint; rupture may be partial or complete

GENETICS

- Unknown
- May be important in increasing the likelihood of active stifle restraint deficiencies and/or conformation abnormalities

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Uncommon in cats

Breed Predispositions

- All susceptible
- Rottweilers and Labrador retrievers—increased incidence when less than 4 years of age

Mean Age and Range

- Dogs, greater than 5 years of age
- Large-breed dogs—1 to 2 years of age

Predominant Sex

- Spayed female

SIGNS/OBSERVED CHANGES in the ANIMAL

- Related to the degree of rupture (partial versus complete), the mode of rupture (sudden [acute] versus long-term [chronic]), the presence of other injury to the stifle, and the severity of inflammation and degenerative joint disease (progressive and permanent deterioration of joint cartilage)
- History of athletic or traumatic events—generally precede sudden (acute) injuries
- Normal activity resulting in sudden (acute) lameness—suggests degenerative rupture; “degeneration” is the decline or loss of function or structure of a tissue
- Subtle to marked intermittent lameness (for weeks to months)—consistent with partial tears that are progressing to complete rupture
- Sudden (acute) cranial cruciate rupture results in non-weightbearing lameness, fluid build-up in the joint (known as “joint effusion”) and the affected leg held in partial flexion while standing
- “Cranial drawer test”—specific manipulation evaluating the status of the cranial cruciate ligament; diagnostic for cranial cruciate rupture
- Decrease in muscle mass (known as “muscle atrophy”) in the rear leg—especially the quadriceps muscle group

CAUSES

- Trauma
- Repetitive micro-injury to the cranial cruciate ligament
- Conformation abnormalities

RISK FACTORS

- Obesity
- Knee cap (known as the “patella”) dislocation (known as a “patellar luxation”)
- Poor conformation
- Abnormalities of the bones making up the stifle



TREATMENT

HEALTH CARE

- Dogs less than 33 lbs (15 kg)—may treat conservatively as outpatients; 65% improve or are normal by 6 months
- Dogs greater than 33 lbs (15 kg)—treat with stabilization surgery; only 20% improve or are normal by 6 months with conservative medical management
- Following surgery—ice packing and physical therapy (such as range-of-motion exercises, massage, and muscle electrical stimulation); important for improving mobility and strength

ACTIVITY

- Restricted—with conservative medical treatment and immediately after surgical stabilization; duration of activity restriction depends on method of treatment and progress of patient

DIET

- Weight control—important for decreasing stress on the stifle joint

SURGERY

- Stabilization surgery—recommended for all dogs; speeds rate of recovery; reduces degenerative joint changes; enhances function
- Various surgical techniques are available to treat cranial cruciate rupture

Extra-Articular Methods

- Variety of techniques that use a heavy-gauge implant to tether the tibia to the femur and restore stability
- Implant material—placed in the approximate plane of the cranial cruciate ligament attachments to the bones (femur and tibia)

Intra-Articular Methods

- Designed to replace the cranial cruciate ligament anatomically
- Uses various materials to “act” as the ligament, including autografts (patella ligament, fascia), allografts (bone-tendon-bone), and synthetic materials

Modified Extra-Articular Methods

- Fibular head transposition or popliteal tendon transposition
- Realignment and tension on the lateral collateral ligament or popliteal tendon to stabilize the stifle joint

Tibial Plateau Leveling Osteotomy (“TPLO”)

- Surgical cutting (known as a “rotational osteotomy”) of the tibia
- Held in place with a special plate and screws

Tibial Tuberosity Advancement

- Surgical procedure in which part of the tibia is cut (procedure known as a “tibial crest osteotomy”), crest is held in an advanced position with a cage and plate, bone graft fills the defect
- Active control of cranial tibial displacement is improved which helps stabilize the stifle



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs)—minimize pain; decrease inflammation; examples include meloxicam, carprofen, etodolac, deracoxib
- Medications intended to slow the progression of arthritic changes and protect joint cartilage (known as “chondroprotective drugs”), such as polysulfated glycosaminoglycans, glucosamine, and chondroitin sulfate—may help limit cartilage damage and degeneration; may help alleviate pain and inflammation



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on method of treatment
- Most surgical techniques require 2 to 4 months of rehabilitation

PREVENTIONS AND AVOIDANCE

- Avoid breeding animals with conformational abnormalities

POSSIBLE COMPLICATIONS

- Second surgery may be required in 10% to 15% of cases, because of subsequent damage to the meniscus (a crescent-shaped cartilage located between the femur and tibia in the stifle)

EXPECTED COURSE AND PROGNOSIS

- Regardless of surgical technique, the success rate generally is better than 85%



KEY POINTS

- Regardless of the method of treatment, some degenerative joint disease (progressive and permanent deterioration of joint cartilage) is common
- Approximately 20% to 40% of dogs with cranial cruciate ligament rupture involving one leg will rupture the ligament in the opposite leg at a later date
- Return to full athletic function is possible, but requires considerable rehabilitation

DISEASE CAUSED BY *CRYPTOCOCCUS*, A TYPE OF FUNGUS (CRYPTOCOCCOSIS)



BASICS

OVERVIEW

- A localized or generalized (systemic) fungal infection caused by the environmental yeast, *Cryptococcus*

GENETICS

- No known influence

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Dogs—American cocker spaniels, Great Danes, Doberman pinschers, and boxers
- Cats—Siamese

Mean Age and Range

- Most common at 2 to 7 years of age (dogs and cats)
- May occur at any age; has been seen often in dogs under 6 months of age

Predominant Sex

- Dogs—none
- Cats—males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary depending on organ systems involved
- May have a history of problems for weeks to months
- Sluggishness (lethargy)
- Mild fever—seen in less than 50% of patients

Dogs

- Nervous system signs—seizures, wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”), weakness (known as “paresis”), blindness
- Skin ulceration
- Enlarged lymph nodes (known as “lymphadenopathy”)
- Vomiting and diarrhea
- Lack of appetite (known as “anorexia”)
- Nasal discharge

Cats

- Nasal discharge
- Nervous system signs—seizures, disorientation, and altered sense of balance (known as a “vestibular disorder”)
- Nodular tissue (known as “granulomas”) seen at the nostrils
- Firm swellings over the bridge of the nose
- Increased breathing rate
- Ulcerated, crusty skin lesions on the head
- Enlarged lymph nodes (lymphadenopathy)
- Eye disease

CAUSES

- *Cryptococcus*, an environmental yeast
- Exposure to cryptococcal organisms and inability of the immune system to prevent colonization and invasion of the organisms into body tissues

RISK FACTORS

- Feline leukemia virus (FeLV) or feline immunodeficiency virus (FIV) infection does not appear to increase risk of *Cryptococcus* infection; however, cats infected with FeLV or FIV have higher risk for more extensive disease and higher likelihood of treatment failure than cats that do not have FeLV or FIV infections



TREATMENT

HEALTH CARE

- Outpatient, if stable
- Nervous system signs—may require inpatient supportive care until stable

ACTIVITY

- No restrictions in most cases

DIET

- No special foods
- Cats—nasal blockage influences appetite; encourage them to eat by offering very tasty food
- Patients treated with itraconazole—give medication in fatty food (such as canned food) to improve absorption of the drug

SURGERY

- Remove nodular masses (known as “granulomatous masses”) in the nose and throat to reduce breathing difficulties



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Fluconazole—preferred for involvement of the eyes or central nervous system, because it is water-soluble and able to enter the nervous system better than some other antifungal drugs; most economical drug choice
- Itraconazole capsules—give with a fatty meal to maximize absorption; pellets in the capsule can be mixed with food, as directed by your pet’s veterinarian; mixing the pellets in food has no apparent bad taste to the patient; itraconazole liquid—better absorption on empty stomach
- Terbinafine has been effective in treatment of cats with resistant infections
- Cryptococcal organisms are prone to become resistant to antifungal treatment
- Amphotericin B (administered intravenously)—may be used in dogs and cats that do not respond to fluconazole or itraconazole; monitor blood work (specifically blood urea nitrogen [BUN] and creatinine) closely to avoid permanent kidney damage (kidney damage is a potential side effect of the drug)



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor blood work (liver enzymes) monthly in patients receiving fluconazole or itraconazole (antifungal drugs); monitor blood work (blood urea nitrogen and creatinine) in patients receiving amphotericin B
- Improvement in clinical signs, resolution of lesions, improvement in well being, and return of appetite measure response to treatment
- Serologic tests (blood tests that detect the presence of antigens of a certain disease-causing agent, in this case *Cryptococcus*), known as “capsular antigen titers”—determine response to and duration of treatment; after 2 months of treatment, the titers should decrease substantially, if treatment is effective; if ineffective, try another antifungal medication, because *Cryptococcus* can become resistant to drugs; “antigens” are any substances that induce an immune response; antigens include fungus (as in this case), proteins, viruses, bacteria, and pollen

PREVENTIONS AND AVOIDANCE

- *Cryptococcus* is found throughout the environment and cannot be avoided

POSSIBLE COMPLICATIONS

- Patients with nervous system disease may have seizures and permanent nervous system changes

EXPECTED COURSE AND PROGNOSIS

- Treatment—anticipated duration of treatment is 3 months to 1 year; patients with central nervous system disease may require lifelong maintenance treatment

- Cats also infected with feline leukemia virus (FeLV) or feline immunodeficiency virus (FIV)—have a worse prognosis
- Serologic tests (blood tests that detect the presence of antigens of a certain disease-causing agent, in this case *Cryptococcus*), known as “capsular antigen titers”—measure every 2 months until 6 months after completion of treatment; continue treatment for 2 months after antigen is no longer detectable, if possible; if patient maintains low titers for months after all signs of disease have resolved, continue treatment for at least 3 months after reduction in antigen levels and resolution of clinical signs and try discontinuing treatment; if titers then rise significantly, resume therapy



KEY POINTS

- Cryptococcosis is a long-term (chronic) disease that requires months of treatment
- The disease is not considered zoonotic (“zoonotic diseases” are diseases that can be passed from animals to people), but it is possible that the yeast may be transmitted to people through bite wounds
- The pet was exposed to the *Cryptococcus* from the environment; family members in the same environment could be at increased risk of infection, especially if they are unable to develop a normal immune response (known as being “immunosuppressed”)

CYANOSIS (BLUISH DISCOLORATION)



BASICS

OVERVIEW

- A bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red blood cells

SIGNALMENT/DESCRIPTION of ANIMAL

A variety of conditions may lead to the development of the bluish discoloration known as “cyanosis.” Cyanosis may be seen in any dog or cat when the oxygen levels in the blood drop below a certain level. The following are some of the conditions that may lead to cyanosis:

- Abnormal blood flow in the heart in which blood from the right side of the heart (where the blood normally has low oxygen levels) is diverted to the left side of the heart (where the blood normally has high oxygen levels); this abnormal blood flow is known as a “right-to-left shunt”—dogs: Keeshonds, English bulldogs, and beagles; some cats; generally young animals
- Tracheal collapse in which a portion of the wall of the windpipe caves in—usually young or middle-aged small-breed dogs (such as Pomeranians, Yorkshire terriers, poodles)
- Congenital laryngeal paralysis in which the animal is born with part of the voice box (larynx) paralyzed—young animals; reported in Dalmatians, Bouvier des Flandres, and Siberian huskies
- Acquired laryngeal paralysis in which part of the voice box (larynx) becomes paralyzed—most common in old, large-breed dogs (such as Labrador retrievers, Afghans, setters, and greyhounds)
- Hypoplastic trachea in which the windpipe develops abnormally and is too small—identified in young English bull terriers; occasionally other breeds
- Asthma (cats)—higher incidence reported in Siamese

SIGNS/OBSERVED CHANGES in the ANIMAL

Cyanosis may be caused by problems associated with low oxygen levels in the blood throughout the body or with problems associated with the actual oxygen-carrying part (hemoglobin) of the red blood cell. These problems cause a type of cyanosis known as “central” cyanosis. A different type is “peripheral” cyanosis in which the bluish discoloration is found in one or more limbs of the body due to decreased blood flow and poor delivery of oxygen-carrying blood to the limb(s). The signs one sees are based on the type of cyanosis—central or peripheral.

- The animal with central cyanosis may have high-pitched, noisy breathing (stridor); difficulty breathing (dyspnea); coughing; voice change; periodic weakness; or fainting (syncope)
- The animal with peripheral cyanosis may have weakness or paralysis of the involved limb(s)

CAUSES

Respiratory (Breathing) System

- Larynx (voice box)—paralysis (acquired or congenital); collapse or caving in of part of the larynx; spasm; fluid build up in the tissues of the voice box (edema); trauma; cancer; nodules
- Trachea (windpipe)—collapse or caving in of part of the windpipe; cancer; foreign body; trauma; abnormal development leading to the windpipe being too small
- Lung disease—pneumonia (multiple types such as viral, bacterial, fungal, allergic, aspiration); chronic bronchitis; hypersensitivity bronchial disease (allergic, asthma); chronic dilation of bronchi; cancer; foreign body; lung parasites (such as worms [filarioidea], flukes [Paragonimus], protozoa); bruising of the lungs (pulmonary contusion) or bleeding (hemorrhage) into the lungs; fluid build up due to non-heart related causes, so called “noncardiogenic edema” (examples of causes of noncardiogenic edema: smoke inhalation, snake bite, electric shock); near drowning
- Pleural space is the space between the lungs and the chest wall; problems in the pleural space that may lead to cyanosis include the presence of free air (pneumothorax); infectious diseases (such as bacterial or fungal pleuritis and feline infectious peritonitis [FIP]) leading to the abnormal build up of fluid and/or other inflammatory materials; the presence of milky fluid known as “chyle” that is a combination of lymph fluid and fat droplets (chylothorax); the presence of blood (hemothorax); cancer; trauma
- Thoracic wall or diaphragm—abnormal developmental conditions that are present at birth (congenital conditions) such as problems involving the sac around the heart (pericardial) and/or the diaphragm in which the diaphragm is not complete and allows some of the contents of the abdomen to move into the chest or into the space between the sac surrounding the heart and the heart itself (types of diaphragmatic hernia); trauma (tearing of the diaphragm with some of the contents of the abdomen slipping into the chest [diaphragmatic hernia], fractured ribs); diseases that affect the nerves and muscles of the chest (such as tick paralysis, coonhound paralysis) that prevent normal breathing

Cardiovascular (Heart and Circulation) System

- Congenital defects are abnormalities in the development of the heart and blood vessels that are present at birth; examples include heart defects such as right-to-left shunting patent ductus arteriosus (PDA), ventricular septal defect (VSD), atrial septal defect (ASD); tetralogy of Fallot; truncus arteriosus; double outlet right ventricle; anomalous pulmonary venous return; lack of normal opening (atresia) of the heart valves (aortic or tricuspid or pulmonary valves)
- Acquired diseases are abnormalities that occur through changes in the heart after the birth of the animal—examples include changes in the heart valve between the left atrium and left ventricle (mitral valve disease); changes in the heart muscle itself (cardiomyopathy)

- Build up of fluid between the sac surrounding the heart (pericardium) and the heart itself is known as “pericardial effusion”—pericardial effusion may develop for unidentifiable reasons (so called “idiopathic disease”) or secondary to cancer
- Blood clots in the lungs are known as “pulmonary thromboembolism;” diseases that may lead to these clots include Cushing’s disease or hyperadrenocorticism in which the adrenal glands produce too much steroid; immune-mediated hemolytic anemia in which the body attacks its own red blood cells; protein-losing nephropathy in which the kidneys are unable to prevent the loss of body proteins into the urine; heartworm disease (dirofilariasis)
- Pulmonary hypertension refers to increased blood pressure involving circulation to the lungs; it may develop for unidentifiable reasons (so called “idiopathic disease”) or from right-to-left cardiac shunts
- Abnormalities involving the arteries and veins of the body are known as “peripheral vascular disease”—examples include blood clots in the arteries (arterial thromboembolism) such as seen in cats with heart muscle disease (feline cardiomyopathies); blockages in the veins (venous obstruction); decreased amount of blood pumped by the heart (reduced cardiac output); shock, narrowing or squeezing of the arteries (arteriolar constriction)

Neuromusculoskeletal (Nervous and Muscle) System

- Brainstem problems—inflammation of the brain (encephalitis); trauma to the brain; bleeding (hemorrhage) into or surrounding the brain; cancer; drug or medication-induced depression of breathing (respiratory center) of the brain
- Spinal cord problems—accumulation of excessive fluid (edema) in the spinal cord; trauma; vertebral fractures; disk prolapse
- Neuromuscular problems—overdose of drugs used to cause paralysis during surgery; tick paralysis; botulism; coonhound paralysis; abnormalities of the autonomic nervous system (dysautonomia); immune-mediated disease leading to abnormal passage of nerve impulses to the muscle (myasthenia gravis)

Methemoglobinemia (Abnormal Oxygen-Carrying Molecule in the Red Blood Cell)

- Abnormal development of the hemoglobin present at birth (congenital)—NADH-MR deficiency (dogs)
- Toxicity due to eating (ingesting) certain chemicals that affect the oxygen-carrying molecule (hemoglobin) of the red blood cell—these chemicals include acetaminophen; nitrates; nitrites; phenacetin; sulfonamides; benzocaine; aniline dyes; dapsone



TREATMENT

HEALTH CARE

- Inpatient—immediate diagnostic testing and treatment
- Stabilization therapy (such as administration of oxygen, tapping the chest to remove accumulated fluid [thoracocentesis], making an opening into the windpipe [tracheostomy])—usually started immediately to improve oxygen levels in the blood and before doing diagnostic testing
- Specific therapy for cause of cyanosis—depends on the final diagnosis
- Diseases associated with cyanosis may be life-threatening

ACTIVITY

- Exercise restriction may be required, based on final diagnosis

DIET

- Dietary modification may be required, based on final diagnosis

SURGERY

- Surgical treatment—depends on primary disease process and the extent of heart or lung involvement



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treatment depends on final diagnosis
- Oxygen therapy as soon as possible
- Diuretics (furosemide)—to remove fluid build up (edema) from the lungs
- To treat methemoglobinemia caused by eating acetaminophen, a chemical that affects the oxygen-carrying molecule (hemoglobin) of the red blood cell—acetylcysteine, cimetidine, and ascorbic acid
- “Clot busters” or plasminogen activators (examples, alteplase, streptokinase)—may use for breaking up clots (thrombolysis) in cats with clots located in their aorta (aortic thromboembolism); best administered by experienced clinicians



FOLLOW-UP CARE

PATIENT MONITORING

- Patients in an oxygen cage should be disturbed as infrequently as possible for monitoring
- Assess response to in-hospital treatment—changes in depth and rate of breathing; color of moist tissues of mouth (mucous membranes) should return to a normal pink color if the cause is not a right-to-left shunt and if patient has adequate reserves; measurement of oxygen levels in the blood (pulse oximetry or arterial blood analysis)
- Following discharge, the client should continue to monitor color of moist tissues of the mouth (mucous membrane) and breathing rate and effort and should seek immediate veterinary care if cyanotic condition returns

POSSIBLE COMPLICATIONS

- Obesity—may complicate or exacerbate underlying respiratory or cardiac diseases
- Advanced pregnancy may exacerbate signs because of pressure on the diaphragm and reduced lung expansion
- Fetuses are likely to be harmed or aborted by the low oxygen levels of the blood (hypoxemia) associated with cyanosis

EXPECTED COURSE AND PROGNOSIS

- Depends on final diagnosis
- Advanced lung or airway disease and severe heart disease—poor long-term outlook (prognosis)



KEY POINTS

- Cyanosis or a bluish discoloration of the skin and moist tissues (mucous membranes) of the body is caused by inadequate oxygen levels in the red blood cells
- Cyanosis is a sign of a potentially life-threatening condition
- Seek immediate veterinary care

DEAFNESS



BASICS

OVERVIEW

- Lack or loss of sense of hearing
- May be complete or partial inability to hear

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- More than 30 breeds of dog have a known susceptibility for deafness (such as the Australian shepherd, Boston terrier, cocker spaniel, Dalmatian, German shepherd dog, Jack Russell terrier, Maltese, toy and miniature poodles, and West Highland white terrier)
- White cats with blue irises; the iris is the colored or pigmented part of the eye

Mean Age and Range

- Congenital (present at birth) deafness—very young age
- Acquired (condition that develops sometime later in life/after birth) deafness—any age; more common in senior dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Deafness involving only one ear (known as “unilateral deafness”) is difficult for most owners to ascertain; thus, most animals brought to clinics for hearing problems have deafness involving both ears (known as “bilateral deafness”)
- Conduction problems in which sound waves do not reach the nerves of hearing (conditions that involve the external ear canal or ear drum may lead to conduction problems)—usually relatively small hearing losses
- Pet does not respond to everyday sounds, does not respond to its name, or cannot be aroused from sleep by a loud noise
- Pet does not respond to the sounds of squeaky toys

CAUSES

Conduction (Sound Waves Do Not Reach the Nerves of Hearing)

- Inflammation of the outer ear (known as “otitis externa”) and other external ear canal disease (such as narrowing of the ear canal, presence of tumors, or ruptured ear drum)
- Inflammation of the middle ear (known as “otitis media”)

Nerve

- Degenerative nerve changes in an old dog
- Anatomic disorders—poor development (known as “hypoplasia”) or lack of development (known as “aplasia”) of the part of the ear (known as the “spiral organ”) that contains the nerve receptors for hearing; condition in which fluid builds up in specific areas of the brain (known as “hydrocephalus” or “water on the brain”) with damage to the part of the brain involved with hearing
- Tumors or cancer involving the nerves of hearing
- Inflammatory and infectious diseases—inflammation of the inner ear (known as “otitis interna”); canine distemper virus (may cause alterations in hearing, not complete deafness); inflammatory masses that develop from the middle ear or eustachian tube (known as “nasopharyngeal polyps”) in cats
- Trauma

Toxins and Drugs

- Antibiotics—aminoglycosides; polymyxin; erythromycin; vancomycin; chloramphenicol
- Antiseptics—alcohol (ethanol); chlorhexidine; cetrimide
- Chemotherapy drugs—cisplatin
- Medications to remove excess fluid from the body (known as “diuretics”)—furosemide
- Heavy metals—arsenic; lead; mercury
- Miscellaneous—products used to break down waxy material in the ear canals (waxy material known as “cerumen;” products known as “ceruminolytic agents”); propylene glycol; salicylates

RISK FACTORS

- Long-term (chronic) inflammation of the outer, middle, or inner ear (otitis externa, media, or interna)
- Merle, piebald gene, or white coat color
- Use of certain drugs



TREATMENT

HEALTH CARE

- Directed toward acquired (condition that develops sometime later in life/after birth) causes
- Congenital (present at birth) deafness is irreversible
- Inflammation of the outer, middle, or inner ear (otitis externa, media, or interna)—medical or surgical approaches depend on extent of disease, bacterial culture and sensitivity test results and X-ray findings
- Conduction problems in which sound waves do not reach the nerves of hearing (conditions that involve the external ear canal or ear drum may lead to conduction problems)—may improve as inflammation of the outer or middle ear (otitis externa or media) resolves
- Hearing aids—have been used; apparatus must be modified for practical use

ACTIVITY

- Restricted to avoid possible injury (for example, a deaf dog cannot hear an approaching car)
- Environment—may need to control for pet's protection

SURGERY

- Surgery may be indicated in cases where the ear canal is narrowed severely or blocked or when a tumor or inflammatory mass that develop from the middle ear or eustachian tube (nasopharyngeal polyp) is present
- Surgery may be indicated in some cases of inflammation of the middle ear (otitis media) to drain the middle ear (procedure known as “bullae osteotomy”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- None specific for deafness
- If present, treat infection or inflammation of the outer, middle, or inner ear (otitis externa, media, or interna)—treatment may include antibiotics, steroids, medications to kill ear mites (*Otodectes*), or medications to treat fungal infections



FOLLOW-UP CARE

PATIENT MONITORING

- Weekly to assess treatment of ear disease until resolved
- Brainstem auditory-evoked response (BAER) test—a test used to evaluate hearing; may be used to monitor response to treatment of inflammation of the inner ear (otitis interna)

POSSIBLE COMPLICATIONS

- Depend on underlying cause

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause



KEY POINTS

- Lack or loss of sense of hearing
- May be complete or partial inability to hear
- Congenital (present at birth) deafness is irreversible

DEMODECTIC MANGE (DEMODICOSIS)



BASICS

OVERVIEW

- An inflammatory parasitic skin disease of dogs and rarely cats, caused by a species of the mite genus, *Demodex*
- Skin disease is characterized by an increased number of mites in the hair follicles and top layer of the skin (known as the “epidermis”), which often leads to secondary bacterial infections (known as “furunculosis”), often with resultant rupturing of the hair follicle
- May be localized (in which one or a few small patches of affected skin are present, frequently seen on the face or forelegs) or generalized (in which numerous skin lesions are present on the head, legs, and body)
- “Demodectic mange,” “demodicosis,” and “red mange” (dogs) are all terms for the same skin disease

GENETICS

- The initial increase in number of demodectic mites in the hair follicles may be the result of a genetic disorder

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and rarely cats

Breed Predilections

- West Highland white terrier—greasy inflammation of the skin with increased accumulations of surface skin cells, such as seen in dandruff (accumulations known as “scales;” condition known as “seborrheic dermatitis”) associated with *Demodex injai*
- Potential of demodectic mange in cats is increased in Siamese and Burmese

Mean Age and Range

- Localized demodectic mange (in which one or a few small patches of affected skin are present, frequently seen on the face or forelegs)—usually in young dogs; median age is 3 to 6 months
- Generalized demodectic mange (in which numerous skin lesions are present on the head, legs, and body)—both young and old dogs
- No age data collected for the cat

SIGNS/OBSERVED CHANGES in the ANIMAL

Dogs

Localized (in which one or a few small patches of affected skin are present, frequently seen on the face or forelegs)

- Lesions—usually mild; consist of reddened skin (known as “erythema”) and a light accumulations of surface skin cells, such as seen in dandruff (scales)
- Patches—several may be noted; most common site is the face, especially around the mouth and eyes; also may be seen on the trunk and legs

Generalized (in which numerous skin lesions are present on the head, legs, and body)

- Can be widespread from the onset, with multiple, poorly circumscribed patches of reddened skin (erythema), hair loss (known as “alopecia”), and accumulations of surface skin cells, such as seen in dandruff (scales)
- As hair follicles become distended with large numbers of mites, secondary bacterial infections (furunculosis) are common, often with resultant rupturing of the hair follicle
- With progression of disease, the skin can become severely inflamed, leading to the escape of fluid and inflammatory cells in or on the skin (known as “exudation”), and the development of nodular, inflammatory lesions (known as “granulomas”)
- *Demodex injai* may be associated with a greasy inflammation of the skin with increased accumulations of surface skin cells, such as seen in dandruff (accumulations are “scales;” condition is “seborrheic dermatitis”) of the dorsal trunk, plugs of keratin and oil in the follicles of the skin (known as “comedones”), reddened skin (erythema), hair loss (alopecia), and darkening of the skin (known as “hyperpigmentation”)

Cats

- Often characterized by multiple partial to complete areas of hair loss (alopecia) of the eyelids, as well as the skin around the eyes, head, neck, flank and the under surface of the body
- Lesions—variable itchiness (known as “pruritus”) with reddened skin (erythema), accumulations of surface skin cells, such as seen in dandruff (scales), and dried discharge on the surface of the skin lesions (known as “crusts”); those caused by *Demodex gato* often are quite itchy (pruritic) and may be contagious
- Inflammation of the outer ear, characterized by the presence of waxy material (known as “ceruminous otitis externa”) has been reported

- *Demodex cati* often is associated with a disease that decreases the immune response (known as “immunosuppressive disease”)

CAUSES

- *Demodex canis*, *Demodex injai*, and an un-named short-bodied demodectic mite
- *Demodex cati* and *Demodex gatoi*

RISK FACTORS

Dogs

- Exact mechanism related to the influence of the immune system on demodectic mange is unknown
- Studies indicate that dogs with generalized demodectic mange (in which numerous skin lesions are present on the head, legs, and body) have a subnormal percentage of interleukin-2 (IL-2) receptors on their lymphocytes and subnormal IL-2 production; “lymphocytes” are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process
- Genetic factors, decreased ability to produce a normal immune response (immunosuppression), and/or metabolic diseases may increase the likelihood that the dog will develop demodectic mange

Cats

- Often associated with metabolic diseases (such as feline immunodeficiency virus [FIV], systemic lupus erythematosus [autoimmune disease in which body attacks its own skin and other organs], diabetes mellitus [sugar diabetes])
- *Demodex gatoi*—may be transferable from cat-to-cat within the same household



TREATMENT

HEALTH CARE

- Outpatient
- Localized demodectic mange (in which one or a few small patches of affected skin are present, frequently seen on the face or forelegs)—conservative; most cases (90%) resolve spontaneously with no treatment
- Generalized demodectic mange (in which numerous skin lesions are present on the head, legs, and body) in dogs—requires application of medication to kill the mites directly onto the skin (known as “topical treatment”) and/or medications administered by mouth (known as “systemic treatment”); antibiotics may be necessary to treat secondary bacterial skin infections
- Evaluate the general health status of dogs with either localized or generalized demodectic mange



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Amitraz (Mitaban®; Taktic EC®)

- A treatment that is applied directly to the skin (topical treatment) to kill demodectic mange mites; agents that kill mites are known as “miticides”
- Use weekly until resolution of clinical signs and no mites are found on skin scrapings; do not rinse off; let air-dry; use as directed by your pet’s veterinarian
- Treat for one month following negative skin scrape
- Apply a benzoyl peroxide shampoo before application of the amitraz to kill bacteria (known as “bactericidal therapy”) and to increase exposure of the mites to the miticide through flushing activity of the hair follicles
- Between 11% and 30% of cases will not be cured; may need to try an alternative therapy or control with maintenance treatment every 2 to 8 weeks
- Rarely used in cats (do not use on diabetic cats)

Ivermectin (Ivomec®; Eqvalan® Liquid)

- Dog—daily administration by mouth has been very effective, even when amitraz fails; use as directed by your pet’s veterinarian
- Treat for 30 to 60 days beyond negative skin scrapings (average length of treatment is 3 to 8 months)
- Reported as a treatment option in the cat, yet the exact dose has not been established

Milbemycin (Interceptor®)

- Has been effective in 50% to 85% of cases
- Treat for 30 to 60 days beyond multiple negative skin scrapings
- Very expensive

Cats

- Exact treatment protocols are not defined
- Lime-sulfur dips applied to the skin (topical treatment) every 3 to 7 days for 4 to 8 treatments often lead to good resolution of clinical signs
- Studies of treatment with ivermectin and milbemycin are lacking, although numerous anecdotal reports suggest effectiveness
- Doramectin also has been reported to be effective when given by injection under the skin (subcutaneous route) once weekly



FOLLOW-UP CARE

PATIENT MONITORING

- Multiple skin scrapings and evidence of resolution of signs

PREVENTIONS AND AVOIDANCE

- Avoid breeding animals with generalized form of demodectic mange (in which numerous skin lesions are present on the head, legs, and body)

POSSIBLE COMPLICATIONS

- Secondary bacterial infections

EXPECTED COURSE AND PROGNOSIS

- Prognosis (dogs)—depends heavily on genetics, status of the immune system, and underlying diseases
- Localized demodectic mange (in which one or a few small patches of affected skin are present, frequently seen on the face or forelegs)—most cases (90%) resolve spontaneously with no treatment; less than 10% of localized demodectic mange cases progress to generalized demodectic mange (in which numerous skin lesions are present on the head, legs, and body)
- Adult-onset of demodectic mange in dogs—often severe disease and poorly responsive to non-responsive to treatment
- Feline cases with *Demodex cati* may have a poor prognosis associated with underlying disease



KEY POINTS

- Localized demodectic mange (in which one or a few small patches of affected skin are present, frequently seen on the face or forelegs)—most cases resolve spontaneously
- Generalized demodectic mange (in which numerous skin lesions are present on the head, legs, and body)—frequent management problem; expense and frustration with the long-term (chronic) nature of disease and treatment are issues; many cases are medically controlled, not cured

DEPIGMENTING DERMATOSES

(SKIN DISORDERS CHARACTERIZED BY LOSS OF PIGMENT)



BASICS

OVERVIEW

- Disease or cosmetic condition involving loss of pigmentation of the skin and/or hair coat
- Normal pigment in the skin and hair coat is melanin

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Mucocutaneous pyoderma (bacterial skin infection involving areas of the lips, eyelids, nostrils)—German shepherd dogs
- Systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs) and discoid lupus erythematosus (autoimmune disease involving the skin only, usually the face)—collies, Shetland sheepdogs, German shepherd dogs
- Pemphigus foliaceus (autoimmune disease involving the skin, characterized by inflammation with crusting and lesions containing pus)—chow chows, Akitas
- Uveodermatologic syndrome (a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris [condition known as “anterior uveitis”] and coexistent inflammation of the skin [known as “dermatitis”], characterized by loss of pigment in the skin of the nose and lips)—Akitas, Samoyeds, Siberian huskies
- Vitiligo (condition characterized by symmetrical lack of pigment in the skin and white hair coat, especially involving the face and nose)—Doberman pinschers and rottweilers; Siamese cats
- Seasonal nasal hypopigmentation (loss of pigment in the tough, hairless skin of the nose [known as the “nasal planum”] that occurs seasonally)—Siberian huskies, Alaskan malamutes, Labrador retrievers
- Proliferative arteritis of the nasal philtrum (inflammation of the arteries of the nasal philtrum, the juncture between the sides of the upper lip extending to the nose)—St. Bernards, giant schnauzers

Mean Age and Range

- Vitiligo (condition characterized by symmetrical lack of pigment in the skin and white hair coat, especially involving the face and nose)—Doberman pinschers and rottweilers, typically less than 3 years of age
- Cutaneous T-cell lymphoma (also known as “mycosis fungoides”)—typically dogs over 10 years of age

Predominant Sex

- Discoid lupus erythematosus (autoimmune disease involving the skin only, usually the face)—may occur more often in females than in males
- Vitiligo in Siamese cats—females

SIGNS/OBSERVED CHANGES in the ANIMAL

- White hair (known as “leukotrichia”)
- Partial or total lack of pigment in the skin (known as “leukoderma”)
- Reddening of the skin (known as “erythema”)
- Loss of the top surface of the skin (known as an “erosion” or “ulceration,” based on depth of tissue loss)

CAUSES

- Mucocutaneous pyoderma (bacterial skin infection involving areas of the lips, eyelids, nostrils)
- Systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs)
- Discoid lupus erythematosus (autoimmune disease involving the skin only, usually the face)
- Pemphigus foliaceus (autoimmune disease involving the skin, characterized by inflammation with crusting and lesions containing pus)
- Pemphigus erythematosus (autoimmune disease involving the skin of the face and ears, characterized by reddening of the skin [erythema] and lesions containing pus)
- Uveodermatologic syndrome (a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris [anterior uveitis] and coexistent inflammation of the skin [dermatitis], characterized by loss of pigment in the skin of the nose and lips)
- Contact hypersensitivity (increased sensitivity or reaction in the skin to the presence of a foreign agent that comes in contact with the skin)
- Vitiligo (condition characterized by symmetrical lack of pigment in the skin and white hair coat, especially involving the face and nose)
- Seasonal nasal depigmentation (loss of pigment in the tough, hairless skin of the nose [known as the “nasal planum”] that occurs seasonally)
- Albinism (inherited disorders characterized by lack of pigment in the skin, hair, and/or eyes, due to abnormal production of melanin)
- Hormonal disorders
- Drug reaction
- Proliferative arteritis of the nasal philtrum (inflammation of the arteries of the nasal philtrum, the juncture between the sides of the upper lip extending to the nose)

- Loss of pigment in the skin and/or hair following skin inflammation
- Dermatophytosis (fungal infection on the surface of the skin)

RISK FACTORS

- Sun exposure—systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs), discoid lupus erythematosus (autoimmune disease involving the skin only, usually the face), and pemphigus erythematosus (autoimmune disease involving the skin of the face and ears, characterized by reddening of the skin [erythema] and lesions containing pus)



TREATMENT

HEALTH CARE

- Outpatient, except for systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs) when severe multiple organ dysfunction is present
- Reduce exposure to sunlight—systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs), discoid lupus erythematosus (autoimmune disease involving the skin only, usually the face) and pemphigus erythematosus (autoimmune disease involving the skin of the face and ears, characterized by reddening of the skin [erythema] and lesions containing pus)
- Replace plastic or rubber dishes—particularly if roughened edges cause abrasions
- Application of water-resistant sun-block ointments or gels (with a SPF greater than 30) to depigmented areas
- Vitiligo and nasal depigmentation—no treatment

ACTIVITY

- Restrict outdoor activity to minimize exposure to sunlight—systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs), discoid lupus erythematosus (autoimmune disease involving the skin only, usually the face) and pemphigus erythematosus (autoimmune disease involving the skin of the face and ears, characterized by reddening of the skin [erythema] and lesions containing pus)

SURGERY

- Skin biopsy



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Vary based on underlying cause
- Systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs)—immunosuppressive therapy with steroids (such as prednisolone or dexamethasone) and chemotherapy drugs (such as azathioprine [dogs] or chlorambucil [cats])
- Medications to decrease the immune response (known as “immunosuppressive therapy”)—to treat systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs), pemphigus foliaceus (autoimmune disease involving the skin, characterized by inflammation with crusting and lesions containing pus), pemphigus erythematosus (autoimmune disease involving the skin of the face and ears, characterized by reddening of the skin [erythema] and lesions containing pus)
- Cyclosporine to decrease the immune response in autoimmune disorders
- Steroids applied to the skin directly (known as “topical steroids”)
- Tacrolimus, 0.1% gel applied daily to lesions in combination with or to replace steroids
- Combination of tetracycline and niacinamide administered by mouth
- Antibiotics for bacterial skin infection (known as “pyoderma”)
- Medications to treat fungal infections (known as “antifungal drugs”) to treat dermatophytosis (fungal infection on the surface of the skin)



FOLLOW-UP CARE

PATIENT MONITORING

- Varies with specific disease and treatment prescribed

PREVENTIONS AND AVOIDANCE

- Restrict outdoor activity to minimize exposure to sunlight—systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs), discoid lupus erythematosus (autoimmune disease involving the skin only, usually the face) and pemphigus erythematosus (autoimmune disease involving the skin of the face and ears, characterized by reddening of the skin [erythema] and lesions containing pus)

POSSIBLE COMPLICATIONS

- Systemic lupus erythematosus (autoimmune disease in which the body attacks its own skin and other organs)—scarring

EXPECTED COURSE AND PROGNOSIS

- Vary with specific disease

ATOPIC DERMATITIS

(INFLAMMATION OF THE SKIN CAUSED BY ALLERGIES)



BASICS

OVERVIEW

- “Atopic” refers to “atopy;” atopy is another name for atopic dermatitis
- “Dermatitis” is inflammation of the skin
- “Allergy” is an altered state of immune response to a foreign substance; “allergen” is a substance to which the animal has developed an allergy
- Atopic dermatitis is a form of skin inflammation due to a susceptibility of the animal to become allergic to normally harmless or innocuous substances, such as pollens (grasses, weeds, and trees), molds, house-dust mites, skin (epithelial) allergens, and other environmental allergens

GENETICS

- Dogs—although an inherited susceptibility is likely, the mode of inheritance is unknown and other factors also may be important
- Cats—unclear

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dogs—any breed, including mixed-breed dogs may be affected; because of probable genetic susceptibility, it may be recognized more frequently in certain breeds or families of dogs, which can vary geographically
- In the United States, commonly affected dog breeds include the Boston terrier, Cairn terrier, dalmatian, English bulldog, English setter, Irish setter, Lhasa apso, miniature schnauzer, pug, Sealyham terrier, Scottish terrier, West Highland white terrier, wire fox terrier, and golden retriever
- Cats—no breed appears to be more likely to develop allergic skin disease (atopic dermatitis)

Mean Age and Range

- Dogs—mean age at onset of signs is 1 to 3 years; range 3 months to 6 years of age; signs may be so mild the first year that they are not observed, but signs usually are progressive and clinically apparent before 3 years of age

Predominant Sex

- Both sexes are probably affected equally

SIGNS/OBSERVED CHANGES in the ANIMAL

- Hallmark sign—“itching” as demonstrated by scratching, rubbing, and/or licking (itchiness is known as “pruritus”)
- Most skin changes probably are produced by self-induced trauma (scratching, rubbing, licking, biting at skin)
- Areas of the face, feet, and under the front legs are affected commonly
- Signs may be seasonal
- Recurring skin and/or ear infections (may be bacterial and/or yeast infection)
- May have temporary response to steroids
- Signs progressively worsen with time
- Lesions—vary from none to broken hairs or saliva discoloration of the hairs (giving a rust-brown appearance to light colored hair) to reddened skin; small, raised skin lesions (known as “papular reactions”); dried discharge on the surface of a skin lesion (known as a “crust”); hair loss (known as “alopecia”); darkened skin (known as “hyperpigmentation”); thickening and hardening of the skin, usually associated with hyperpigmentation (known as “lichenification”); and excessively oily or dry scaling of the skin (known as “seborrhea”)
- Inflammation of the moist tissues around the eye (known as “conjunctivitis”) may occur

CAUSES

- Airborne pollens (grasses, weeds, and trees)
- Mold spores (indoor and outdoor)
- House-dust mites
- Animal danders
- Insects (controversial)

RISK FACTORS

- Temperate environments with long allergy seasons and high pollen and mold-spore levels
- Coexistent skin disorders characterized by itchiness (known as “pruritic dermatoses”), such as flea-bite hypersensitivity and adverse food reaction; these coexistent skin disorders increase the severity of the signs



TREATMENT

HEALTH CARE

- Outpatient
- Frequent bathing in cool water with shampoos designed to minimize itchiness can be beneficial

ACTIVITY

- Avoid substances (allergens) to which the animal is allergic, when possible

DIET

- Diets rich in essential fatty acids may be beneficial in some cases



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Immunotherapy (Hyposensitization or “Allergy Shots”)

- Administration (usually subcutaneous [SC] injections) of gradually increasing doses of the causative allergens to affected patients in an attempt to reduce their sensitivity to the particular substance(s)
- Allergen selection—based on allergy test results, patient history, and knowledge of local plants that contribute pollen into the air
- Indicated when it is desirable to avoid or reduce the amount of steroids required to control signs, when signs last longer than 4 to 6 months per year, or when nonsteroidal forms of therapy are ineffective
- Successfully reduces itchiness (pruritus) in 60% to 80% of dogs and cats
- The response to “allergy shots” is usually slow, often requiring 3 to 6 months and up to 1 year to see response

Steroids

- May be given for short-term relief and to break the “itch-scratch cycle”
- Should be tapered to the lowest dosage that adequately controls itchiness (pruritus), as directed by your pet’s veterinarian
- Best choices—prednisolone or methylprednisolone tablets
- Cats may need methylprednisolone acetate treatment, administered by injection

Antihistamines

- Less effective than are steroids
- Evidence of effectiveness is poor
- Dogs—antihistamines include hydroxyzine, chlorpheniramine, diphenhydramine, and clemastine
- Cats—chlorpheniramine; effectiveness estimated at 10% to 50%

Other Medications

- Tricyclic antidepressants (“TCAs,” such as doxepin or amitriptyline) have been given to dogs to control itchiness, but their overall effectiveness and mode of action is unclear; not extensively studied in the cat
- Cyclosporine (Atopica®) is effective in controlling itchiness (pruritus) associated with long-term (chronic) allergic skin disease (atopic dermatitis); the response is variable—many patients can be controlled adequately long-term with less frequent dosing (such as every 2 to 4 days), as directed by your pet’s veterinarian; frequent patient monitoring is recommended
- Topical triamcinolone spray 0.015% (Genesis®, Virbac) can be applied to the skin over large body surfaces to control itchiness (pruritus) with minimal side effects



FOLLOW-UP CARE

PATIENT MONITORING

- Examine patient every 2 to 8 weeks when a new course of treatment is started
- Monitor itchiness (pruritus); self-trauma, such as scratching or licking; skin infection characterized by the presence of pus (known as “pyoderma”); and possible adverse drug reactions
- Once an acceptable level of control is achieved, examine patient every 3 to 12 months
- A complete blood count (CBC), serum chemistry profile, and urinalysis—recommended every 3 to 12 months for patients on long-term (chronic) steroid or cyclosporine therapy

PREVENTIONS AND AVOIDANCE

- If the substances (allergens) to which the animal is allergic have been identified through allergy testing, the owner should undertake to reduce the animal's exposure to these substances, as much as possible
- Minimizing other sources of itchiness ([pruritus], such as fleas, adverse food reactions, and secondary skin infections) may reduce the level of pruritus enough to be tolerated by the animal

POSSIBLE COMPLICATIONS

- Secondary skin infection characterized by the presence of pus (pyoderma) or inflammation of the skin due to yeast (*Malassezia* dermatitis)
- Coexistent flea-bite allergy (hypersensitivity) and/or adverse food reaction

EXPECTED COURSE AND PROGNOSIS

- Not life-threatening, unless itchiness (pruritus) is not responsive to medical treatment and it is so disruptive that the result is euthanasia
- If left untreated, the degree of itchiness (pruritus) worsens and the duration of signs last longer each year of the animal's life
- Some cases may resolve spontaneously



KEY POINTS

- Atopic dermatitis is a progressive skin condition
- It rarely goes into remission and cannot be cured
- Some form of therapy may be necessary for life to control the signs (itchiness, rubbing, scratching)

DERMATOMYOSITIS

(INFLAMMATION OF THE SKIN AND MUSCLES)



BASICS

OVERVIEW

- “Dermatomyositis” is an inherited inflammatory disease of the skin, muscles, and blood vessels that develops in young collies, Shetland sheepdogs, and their crossbreeds
- Similar signs have been reported in other breeds, such as the Beauceron shepherd, Welsh corgi, Lakeland terrier, chow chow, German shepherd dog, and Kuvasz, as well as individual dogs; however, the condition in these dogs currently is classified as “ischemic dermatopathy” and not “dermatomyositis” as previously reported

GENETICS

- Collies and Shetland sheepdogs—studies suggest that dermatomyositis is inherited in an autosomal dominant manner, with variable expression

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Collies, Shetland sheepdogs, and their crossbreeds

Mean Age and Range

- Skin lesions typically develop before six months of age, and may develop as early as 7 weeks of age
- The full extent of lesions usually is present by 1 year of age, and may lessen thereafter
- Adult-onset dermatomyositis can occur, but is rare

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs vary from subtle skin lesions and inflammation of muscles that does not cause clinical signs (known as “subclinical myositis”) to severe skin lesions and a generalized decrease in muscle mass (known as “muscle atrophy”) with an enlarged esophagus (part of the digestive tract, the tube running from the throat to the stomach; condition known as “megaesophagus”)
- Skin lesions around the eyes, lips, face, inner surface of the prick ears, tip of the tail, and bony prominences vary in intensity; the entire face may be involved—skin lesions may increase and decrease over time (known as a “waxing and waning” course); signs usually seen in affected dogs before they are 6 months of age
- Skin lesions—characterized by variable degrees of crusted areas with loss of the top surface of the skin (known as “erosions” or “ulcers”, based on depth of tissue loss) and hair loss (known as “alopecia”), with reddening of the skin (known as “erythema”), accumulations of surface skin cells, such as seen in dandruff (known as “scales”), and scars
- Scars may occur as a sequela to initial skin lesions
- More severely affected dogs may have difficulty eating, drinking, and swallowing
- Several litter mates may be affected, but the severity of the disease often varies significantly among affected dogs
- Foot-pad ulcers and ulcers in the mouth, as well as nail abnormalities or loss, may occur
- Inflammation of the muscles (myositis)—signs may be absent or vary from subtle decrease in the mass of the muscles extending from the top and side of the head, behind the eye, to the lower jaw (known as the “temporal muscles”) to generalized, symmetric loss of muscle mass (muscle atrophy) and stiff gait
- Decrease in muscle mass (muscle atrophy) of the muscles extending from the bone below the eye to the lower jaw (known as the “masseter muscles”) that act to close the jaw and muscles extending from the top and side of the head, behind the eye, to the lower jaw (temporal muscles) that act to close the jaw—may be evident
- Dogs with enlarged esophagus (megaesophagus) may present with aspiration pneumonia

CAUSES

- Hereditary
- Infectious agents
- Immune-mediated disease

RISK FACTORS

- Mechanical pressure and trauma, and ultraviolet-light exposure may worsen skin lesions



TREATMENT

HEALTH CARE

- Most dogs can be treated as outpatients
- Dogs with severe inflammation of the muscles (myositis) and enlarged esophagus (megaesophagus) may need to be hospitalized for supportive care
- Euthanasia may be indicated in severe cases

ACTIVITY

- Avoid activities that may traumatize the skin
- Keep indoors during the day to avoid exposure to intense sunlight

DIET

- May need to change diet, if dog has enlarged esophagus (megaesophagus) or has difficulty eating and/or swallowing

SURGERY

- Skin biopsy—may be diagnostic for dermatomyositis, although this disease can be difficult to diagnose definitively
- Muscle biopsy



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonspecific symptomatic therapy includes hypoallergenic shampoo baths, treating secondary bacterial skin infection (known as “pyoderma”) and *Demodex* mange (known as “demodicosis”), and avoiding trauma and sunlight
- Vitamin E
- Essential fatty acid supplements
- Steroids (such as prednisone) to decrease inflammation
- Pentoxifylline to improve blood flow and to reduce inflammation



FOLLOW-UP CARE

PREVENTIONS AND AVOIDANCE

- Do not breed affected animals
- Neuter intact animals
- Minimize trauma and exposure to sunlight

POSSIBLE COMPLICATIONS

- Secondary bacterial skin infection (pyoderma) and *Demodex* mange (demodicosis)
- Mildly to moderately affected dogs may have residual scarring
- Severely affected dogs may have trouble chewing, drinking, and swallowing
- Enlarged esophagus (megaesophagus) may develop, increasing the likelihood of aspiration pneumonia

EXPECTED COURSE AND PROGNOSIS

- The effectiveness of medical treatment can be difficult to assess because the disease tends to be cyclic in nature and often is

self-limiting

- Long-term prognosis—variable, depending on severity of disease
- Minimal disease—prognosis good; tends to resolve spontaneously with no evidence of scarring
- Mild to moderate disease—tends to resolve spontaneously, but residual scarring is common
- Severe disease—prognosis for long-term survival is poor as the inflammation of the skin (known as “dermatitis”) and muscles (myositis) may be lifelong



KEY POINTS

- Dermatomyositis is considered an inherited disease
- Affected dogs should not be used for breeding
- The disease is not curable, although spontaneous resolution or waxing and waning of signs may occur

FUNGAL INFECTION OF THE SKIN, HAIR OR NAILS (DERMATOPHYTOSIS)



BASICS

OVERVIEW

- “Dermatophytosis” is the medical term for a fungal infection affecting the skin, hair, and/or nails (claws)
- Most commonly isolated fungal organisms are *Microsporum canis*, *Trichophyton mentagrophytes*, and *Microsporum gypseum*

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs, cats, other mammals

Breed Predilections

- In cats, infections are seen more commonly in long-haired breeds

Mean Age and Range

- Clinical signs are seen more commonly in young animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Pet may be an inapparent carrier; a “carrier” is an animal in which no signs of disease are present, but harbors the disease-causing fungus and can transmit it to other animals or people
- Hair loss (known as “alopecia”), which may be patchy or circular; the classic sign of circular hair loss is more common in cats than in dogs
- Poor hair coat
- Scales (accumulations of surface skin cells, such as seen in dandruff); reddened skin (known as “erythema”); darkened skin (known as “hyperpigmentation”); and itchiness (known as “pruritus”) are variable
- Inflammation of the claw folds (known as “paronychia”), nodular lesions (known as “granulomatous lesions”), or raised nodular lesions that frequently ooze (known as “kerions”) also may be seen

CAUSES

- *Microsporum canis* is by far the most common cause of dermatophytosis in cats
- In dogs, the three most common causes are *Microsporum canis*, *Microsporum gypseum*, and *Trichophyton mentagrophytes*; the incidence of each fungus varies geographically
- Less common species can cause fungal infection of the skin, hair, and/or nails (dermatophytosis)

RISK FACTORS

- Diseases or medications that decrease the ability of the body to develop a normal immune response (known as “immunocompromising diseases” or “immunosuppressive medications,” respectively) increase the likelihood that a pet will develop a fungal infection of the skin, hair, and/or nails (dermatophytosis) and increase the potential for a more severe infection
- High population density of animals (for example, in a cattery or animal shelter), poor nutrition, poor management practices, and lack of adequate quarantine period increase risk of infection



TREATMENT

HEALTH CARE

- Most pets are treated as outpatients
- Quarantine procedures should be considered due to the infective and zoonotic nature of the disease; “zoonotic diseases” are diseases that can be passed from animals to people
- The use of an Elizabethan collar, particularly in cats, is recommended to prevent ingestion of antifungal medications applied to the skin
- A “ringworm vaccine” was available, but apparently it was only of benefit in decreasing signs, which might lead to development of a carrier (an animal with no signs of disease, but which harbors the disease-causing fungus and can transmit it to other animals or people); the vaccine is no longer available in the United States

ACTIVITY

- Within limits of quarantine, physical activity can remain as normal as possible

DIET

- Depending on the medication used in treatment, the diet should remain normal
- If griseofulvin (an antifungal drug) is used as treatment, a fatty meal improves absorption following administration of the drug by mouth



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Griseofulvin (an antifungal drug) has been prescribed most commonly for the treatment of dermatophytosis; griseofulvin's absorption is enhanced by dividing the dose twice per day or giving it with a fatty meal; griseofulvin does have side effects, some of which are serious—discuss potential side effects with your pet's veterinarian
- Ketoconazole (an antifungal drug) has shown effectiveness in the treatment of dermatophytosis; treatment usually requires 4 to 8 weeks; side effects (such as lack of appetite, vomiting, and liver disease) have been seen—discuss potential side effects with your pet's veterinarian
- Itraconazole is similar to ketoconazole, but typically has fewer side effects and is likely more effective; treatment usually requires 4 to 8 weeks
- Clipping of the hair coat and application of antifungal medications directly to the skin (known as “topical therapy”) may be used in treatment; topical treatments often are associated with initial worsening of signs; topical treatments include lime sulfur (1:16 dilution or 8 oz per gallon of water), enilconazole and miconazole (with or without chlorhexidine)
- Lufenuron, a chitin-synthesis inhibitor used in flea control, was once a popular treatment consideration, but studies have suggested inconsistent results
- Fluconazole (an antifungal drug) is an alternative treatment that is largely untested and offers no benefit over itraconazole; it is likely even less effective



FOLLOW-UP CARE

PATIENT MONITORING

- Fungal (dermatophyte) culture is the only means of truly monitoring response to treatment
- Many animals will improve clinically, but remain fungal culture positive
- It is advisable to repeat fungal cultures toward the end of treatment and continue treatment until at least one culture result is negative
- In resistant cases, fungal cultures may be repeated on a weekly basis and treatment continued until 2 to 3 consecutive negative results are obtained
- Complete blood counts should be performed weekly or biweekly for animals receiving griseofulvin
- Blood work to monitor liver changes may be indicated for animals receiving ketoconazole or itraconazole

PREVENTIONS AND AVOIDANCE

- The use of a quarantine period and fungal (dermatophyte) cultures of all animals entering the household are necessary to prevent reinfection from other animals
- The possibility of rodents aiding in the spread of the disease should be considered
- Treatment of exposed animals can be considered to prevent development of clinical signs

POSSIBLE COMPLICATIONS

- Falsely negative fungal (dermatophyte) cultures complicate management of this disease

EXPECTED COURSE AND PROGNOSIS

- Many animals will self clear a fungal infection of the skin, hair, and/or nails (dermatophytosis) over a period of a few months
- Treatment hastens clinical cure and helps reduce environmental contamination
- Some infections, particularly in long-haired cats or multi-animal homes or facilities, can be very persistent



KEY POINTS

- Many dogs and short-haired cats (in a single cat environment) will undergo spontaneous remission
- The treatment of fungal infection of the skin, hair, and/or nails (dermatophytosis) can be frustrating and expensive, especially in multi-animal households or facilities or in recurrent cases
- Environmental treatment is not pursued as often as it probably should be, especially in recurrent cases; dilute bleach (1:10) is a practical and relatively effective means of providing environmental decontamination; however, this dilution of bleach will bleach

various household materials—discuss the use of bleach in the environment with your pet’s veterinarian

- In a multi-animal environment or cattery situation, treatment and control of this disease can be very complicated
- Dermatophytosis is a zoonotic disease; “zoonotic diseases” are diseases that can be passed from animals to people
- If a person in contact with a dog or cat develops skin lesions, they should seek medical attention

EXFOLIATIVE DERMATOSES

(SKIN DISORDERS CHARACTERIZED BY THE PRESENCE OF SCALES)



BASICS

OVERVIEW

- Excessive or abnormal shedding of skin cells, resulting in the clinical presentation of accumulations of surface skin cells, such as seen in dandruff (known as “scales”)
- “Exfoliative” refers to the detachment and shedding of surface skin cells; “dermatosis” (plural, “dermatoses”) is the medical term for any skin abnormality or disorder
- “Primary” refers to a condition that occurs first in the skin; “secondary” refers to changes that occur following the primary disease—for example, the skin may be inflamed due to a nutritional deficiency (primary condition) and become infected by bacteria invading the inflamed skin (secondary condition) or the skin changes are secondary to the presence of some other problem or abnormality (such as the presence of parasites on the skin)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Primary exfoliative dermatoses—cocker spaniels, English springer spaniels, West Highland white terriers, basset hounds, Doberman pinschers, Irish setters, Labrador retrievers, Siberian huskies, Alaskan malamutes, dachshunds, chow chows, Yorkshire terriers, poodles, Great Danes, whippets, salukis, Italian greyhounds, standard poodles, Samoyeds, Akitas, vizslas, golden retrievers
- Secondary exfoliative dermatoses—any breed of dog or cat

Mean Age and Range

- Primary exfoliative dermatoses—apparent by 2 years of age
- Secondary exfoliative dermatoses—any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Excessive scaling
- Smelly or malodorous skin; “rancid fat” odor is common
- Itchiness (known as “pruritus”)
- Dry or greasy accumulations of surface skin cells, as seen in dandruff (scales); accumulations may be fine or coarse; may be located throughout the hair coat or in localized areas
- Hair follicles may become filled with oil and skin cells (known as “comedones”)
- Accumulation of debris that adheres to hair shaft (known as “follicular casts”)
- Hair loss (known as “alopecia”)
- Secondary skin infection characterized by the presence of pus (known as “secondary pyoderma”)

CAUSES AND RISK FACTORS

Primary Exfoliative Dermatoses

- Primary excessively dry or oily scaling of the skin (known as “seborrhea”) of unknown causes (so called “idiopathic seborrhea”) that is a primary disorder in the normal replacement and shedding of skin cells (known as a “keratinization disorder”)—breeds at highest risk: cocker spaniels, English springer spaniels, West Highland white terriers, basset hounds, Doberman pinschers, Irish setters, and Labrador retrievers; dry (known as “seborrhea sicca”) and greasy or oily (known as “seborrhea oleosa”) forms exist, but determination of type has little prognostic value
- Skin disorder that responds to treatment with vitamin A (known as “vitamin A-responsive dermatosis”)—nutritionally responsive; seen primarily in young cocker spaniels; clinical signs similar to severe idiopathic seborrhea; distinguished by response to dietary vitamin A supplementation
- Skin disorder that responds to treatment with zinc (known as “zinc-responsive dermatosis”)—nutritionally responsive; results in hair loss (alopecia); accumulations of surface skin cells, as seen in dandruff (scales); dried discharge on the surface of the skin lesion (known as a “crust”); and reddening of the skin (known as “erythema”) around the eyes, ears, feet, lips, and other external orifices; two syndromes are seen: 1) young adult dogs, especially Siberian huskies and Alaskan malamutes and 2) rapidly growing, large-breed puppies
- Abnormalities in the development of the skin and related structures (such as hair follicles)—abnormal development of the hair follicles or hair (known as “follicular dysplasia”); seen as hair loss (alopecia) in color mutant or dilution animals; represent abnormalities in deposition of melanin pigments (responsible for the color of the skin and hair) of the hair shaft and structural hair growth; breeds commonly affected: blue and fawn Doberman pinschers, Irish setters, dachshunds, chow chows, Yorkshire terriers, poodles, Great Danes, whippets, salukis, and Italian greyhounds; signs include failure to regrow blue or fawn hair with normal “point” hair growth, excessive accumulations of surface skin cells, as seen in dandruff (scales), hair follicles filled with oil and skin cells (comedones), and skin infection characterized by the presence of pus (secondary pyoderma)
- Thickening of the skin (known as “hyperkeratosis”) of the nose and pads of the feet of unknown cause (so called “idiopathic nasodigital hyperkeratosis”)—excessive accumulation of surface skin cells (scales) and dried discharge on the surface of the skin lesion (crusts) on the tough, hairless skin of the nose (known as the “nasal planum”) and footpad margins; common in middle-aged spaniels; if

severe, may result in cracking and secondary bacterial infection

- Inflammation of the sebaceous glands, the glands that produce oils in the hair coat (condition known as “sebaceous adenitis”)—inflammatory disease; may be of unknown cause (so called “idiopathic sebaceous adenitis”); three specific syndromes seen: 1) middle-aged standard poodles and Samoyeds; characteristic widespread (diffuse) hair loss (alopecia) and excessive accumulation of surface skin cells, as seen in dandruff (scales); accumulation of debris that adheres to hair shaft (follicular casts); most dogs generally appear healthy; 2) Akitas: frequently develop severe and deep bacterial skin infection characterized by the presence of pus (pyoderma); 3) vizslas: disease appears distinctly different and is characterized by the presence of nodular, inflammatory lesions (known as “granulomas”)
- Abnormal development of the top surface of the skin (known as the “epidermis,” condition known as “epidermal dysplasia”) and congenital (present at birth) disorders of the normal replacement and shedding of skin cells (keratinization disorder; condition known as “ichthyosis”)—rare and severe congenital disorder of keratinization; reported in West Highland white terriers and golden retrievers; generalized accumulations of surface skin cells, as seen in dandruff (scales) and dried discharge on the surfaces of the skin lesions (crusts) at an early age; secondary bacterial and yeast infections are common

Secondary Exfoliative Dermatoses

- Increased sensitivity or reaction in the skin to the presence of a foreign agent (known as “cutaneous hypersensitivity”)—atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems); flea-bite allergy; food allergy; and inflammation of the skin secondary to contact with some substance to which the animal reacts or to which the skin becomes irritated (known as “contact dermatitis”); characterized by itchiness (pruritus) and resultant skin trauma and irritation
- Parasites of the skin—sarcoptic mange or scabies; demodectic mange (demodicosis), and “walking dandruff” (cheyletiellosis)
- Skin infection characterized by the presence of pus (pyoderma)
- Skin inflammation due to *Malassezia*, a yeast
- A fungal infection affecting the skin, hair, and/or nails (known as “dermatophytosis”)
- Hormonal disorders (known as “endocrinopathies”)—inadequate production of thyroid hormone (known as “hypothyroidism”) and excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”) commonly produce excessive accumulation of surface skin cells, as seen in dandruff (scales); secondary skin infection characterized by the presence of pus (secondary pyoderma) common in both syndromes; other hormonal abnormalities (such as sex-hormone abnormalities, excessive production of thyroid hormone [known as “hyperthyroidism”], and diabetes mellitus [“sugar diabetes”]) also may be associated with excessive scaling
- Age—senior animals may have a dull, brittle, and scaly hair coat; changes may be caused by natural alterations associated with aging; no specific defect identified
- Nutritional disorders—skin disorders may occur with malnutrition and feeding generic dog food; result in accumulation of surface skin cells, as seen in dandruff (scales) from abnormalities in the normal replacement and shedding of skin cells (keratinization)
- Diseases in which the body’s immune system attacks its own skin (known as “autoimmune skin diseases”)—pemphigus complex; cutaneous and systemic lupus erythematosus
- Tumors, cancer, and precancerous disorders of the skin—may produce hair loss (alopecia) and accumulation of surface skin cells, as seen in dandruff (scales) when skin is damaged
- Miscellaneous—any disease process may result in excessive accumulation of surface skin cells, as seen in dandruff (scales)



TREATMENT

HEALTH CARE

- Diagnose and control all treatable primary and secondary diseases
- Frequent and appropriate treatment applied to the skin directly (known as “topical therapy”)—cornerstone of proper treatment
- Frequent baths, as directed by your pet’s veterinarian
- Recurrence of secondary infections may require repeated treatment and further diagnostic testing
- Maintaining control of these skin disorders is often a lifelong commitment

DIET

- Depends on underlying cause
- Dietary modification may be necessary for cases of suspected food allergy and for nutritionally related skin disorders

SURGERY

- Skin biopsy may be necessary to determine diagnosis
- Surgical removal of skin tumors/cancer



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Treatment Applied to the Skin Directly (Topical Therapy)

Shampoos

- Contact time of shampoo (that is, shampoo must be left on body for a certain amount of time)—5 to 15 minutes generally required, as directed by your pet's veterinarian
- Hypoallergenic ("soap-free") shampoo—useful only in mild cases of dry scale and to maintain skin condition after the primary disease has been controlled
- Sulfur/salicylic acid-containing shampoos—soften and loosen crusts and scales on the skin (known as "keratolytic shampoos") and slow the growth of bacteria (known as a "bacteriostatic property"); excellent first choice for the moderately scaly patient; not overly drying
- Benzoyl peroxide shampoo—strong action to soften and loosen crusts and scales on the skin (keratolytic shampoo), destroys bacteria or slows their growth and multiplication (known as an "antimicrobial property"), and flushes out the hair follicles; may cause irritation and severe dryness; frequently used for recurrent bacterial infection and/or extreme greasiness
- Ethyl lactate shampoo—less effective than benzoyl peroxide for flushing out the hair follicles and not as effective against bacteria, but not as irritating or drying; most useful for moderate skin infection characterized by the presence of pus (pyoderma) and dry scale
- Tar shampoos—soften and loosen crusts and scales on the skin (keratolytic shampoos) and decreases itchiness (known as an "antipruritic property"); degreasing, but less so than benzoyl peroxide; use for moderate scale associated with itchiness (pruritus)

Moisturizers

- Excellent for restoring skin hydration (frequent shampooing may result in excessive dryness and discomfort) and increasing effectiveness of subsequent shampoos
- Humectants (moisturizers that attract water into the surface of the skin)—encourage hydration of the skin; at high concentrations may soften and loosen crusts and scales on the skin
- Microencapsulation (moisturizers are placed in tiny capsules that allow prolonged effect)—recent advances may improve the residual activity of moisturizers by permitting sustained release after bathing
- Emollients (agents that soften and soothe the skin)—coat the skin; smooth the roughened surfaces produced by excessive scaling

Generalized (Systemic) Therapy

- Specific causes of exfoliative dermatoses require specific treatments (such as thyroid hormone [thyroxine] replacement for animals with inadequate levels of thyroid hormone [hypothyroidism]; zinc supplements for zinc-responsive dermatosis)
- Antibiotics administered by mouth or injection—always indicated for secondary skin infection characterized by the presence of pus (secondary pyoderma)
- Retinoid drugs—varied success for seborrhea of unknown cause (idiopathic seborrhea) or primary seborrhea; reports of individual response to retinoids (especially cocker spaniels with a primary keratinization defect); generally, topical therapy provides more benefits for dogs than does retinoid administration
- Cyclosporine (medication to decrease the immune response) may be used in some cases
- Ketoconazole (an antifungal drug)—may be used for treatment of severe skin inflammation due to *Malassezia*, a yeast



FOLLOW-UP CARE

PATIENT MONITORING

- Antibiotics and treatment applied to the skin directly (topical therapy)—recheck every 3 weeks to monitor response; patients may respond differently to the various topical therapies
- Seasonal changes, development of additional diseases (especially increased sensitivity or reaction in the skin to the presence of a foreign agent [cutaneous hypersensitivity]), and recurrence of skin infection characterized by the presence of pus (pyoderma)—may cause previously controlled patients to worsen; re-evaluation critical for determining if new factors are involved and if changes in therapy are necessary
- Hormonal disorders (endocrinopathies)—blood work to monitor thyroid levels following treatment for inadequate levels of thyroid hormone (hypothyroidism), should be performed 4 to 6 hours following administration of thyroid medication—frequency of thyroid monitoring as recommended by your pet's veterinarian; ACTH-stimulation tests should be performed for proper management of pets with excessive production of steroids by the adrenal glands (hyperadrenocorticism or Cushing's disease)
- Some autoimmune disorders—re-evaluate frequently during the initial phase of treatment; less often after remission
- Medications to decrease the immune response (known as "immunosuppressive therapy")—frequent blood work (such as complete blood counts [CBCs] and serum chemistries) and urinalyses with bacterial culture to monitor for complications
- Retinoid drugs—blood work (serum chemistries, including triglycerides); Schirmer tear tests to monitor the eyes for changes in tear production
- Ketoconazole—blood work (serum chemistries)

PREVENTIONS AND AVOIDANCE

- Depend on underlying cause

POSSIBLE COMPLICATIONS

- Depend on underlying cause

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause



KEY POINTS

- Some causes of excessive or abnormal shedding of skin cells, resulting in the clinical presentation of accumulations of surface skin cells (such as dermatophytosis and several parasitic skin diseases) have either zoonotic potential or the ability to produce human lesions; “potential zoonoses” are diseases that can be passed from animals to people
- Diagnose and control all treatable primary and secondary diseases
- Frequent and appropriate treatment applied to the skin directly (known as “topical therapy”)—cornerstone of proper treatment
- Frequent baths, as directed by your pet’s veterinarian
- Recurrence of secondary infections may require repeated treatment and further diagnostic testing
- Maintaining control of these skin disorders is often a lifelong commitment

SKIN DISORDERS OF THE NOSE (NASAL DERMATOSES)



BASICS

OVERVIEW

- Conditions characterized by abnormalities of the skin on the nose, involving either the haired portion (bridge of the nose) or non-haired portion (known as the “nasal planum”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Zinc-responsive skin disorder (known as “zinc-responsive dermatosis”)—Siberian huskies, Alaskan malamutes
- Inflammatory disorder that affects the skin and muscles of unknown cause (condition known as “idiopathic dermatomyositis”)—collies and Shetland sheepdogs
- Syndrome in which the animal has inflammation in the front part of the eye, including the iris (known as “anterior uveitis”) and coexistent inflammation of the skin (known as “dermatitis”), characterized by loss of pigment in the skin of the nose and lips (known as “uveodermatologic syndrome”)—Akitas, Samoyeds, Siberian huskies
- Systemic lupus erythematosus and discoid lupus erythematosus (autoimmune diseases that involve the skin)—collies, Shetland sheepdogs, German shepherd dogs

Mean Age and Range

- Fungal infection (known as “dermatophytosis”) of the nose, zinc-responsive skin disorder (zinc-responsive dermatosis), inflammatory disorder that affects the skin and muscles (dermatomyositis), and demodectic mange (known as “demodicosis”)—more likely in dogs less than 1 year of age
- Epidermotropic lymphoma (skin cancer that develops from lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body)—old dogs

Predominant Sex

- Discoid lupus erythematosus (autoimmune disease that involves the skin) may occur more often in females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Loss of pigment of the hair and/or skin of the nose (known as “depigmentation”)
- Darkening or increased pigment of the skin (known as “hyperpigmentation”)
- Reddening of the skin (known as “erythema”)
- Loss of the top layer of the skin of the nose (known as an “erosion” or “ulceration”)
- Blister or small, circumscribed elevation of the outer layer of the skin filled with clear fluid (known as a “vesicle”) or small, circumscribed elevation of the outer layer of the skin filled with pus (known as a “pustule”)
- Dried discharge on the surface of a skin lesion (known as a “crust”)
- Scarring
- Loss of hair (known as “alopecia”)
- Small, solid masses (known as “nodules”) and/or thickened, raised, flat-topped areas that are slightly higher than the normal skin (known as “plaques”)

CAUSES

- Skin infection of the nose, characterized by the presence of pus (known as “nasal pyoderma”)
- Demodectic mange (demodicosis)
- Fungal skin infection (dermatophytosis)
- Deep fungal infections—cryptococcosis, sporotrichosis, aspergillosis
- Immune-mediated disease—examples include the following autoimmune diseases that involve the skin: systemic lupus erythematosus, discoid lupus erythematosus, pemphigus foliaceus, pemphigus erythematosus
- Inflammation of the skin in the lightly pigmented skin of the nose secondary to exposure to sunlight (known as “nasal solar dermatitis”)

- Inflammatory disorder that affects the skin and muscles (dermatomyositis)—collies and Shetland sheepdogs
- Zinc-responsive skin disorder (zinc-responsive dermatosis)
- Inflammation in the front part of the eye, including the iris (anterior uveitis) and coexistent inflammation of the skin (dermatitis), characterized by loss of pigment in the skin of the nose and lips (uveodermatologic syndrome)
- Condition characterized by symmetrical lack of pigment in the skin and white hair coat, especially involving the face and nose (known as “vitiligo”)
- Loss of pigment of the hair and/or skin of the nose (nasal depigmentation)
- Inflammation of the skin resulting from contact with some offending substance (known as “contact hypersensitivity”)—inflammation of the skin of the nose secondary to contact with plastic food bowls (known as “plastic-dish dermatitis”), skin inflammation secondary to application of medication onto the nose (known as “topical drug hypersensitivity;” the antibiotic, neomycin, frequently is involved)
- Tumors or cancer—types include the following: squamous cell carcinoma, basal cell carcinoma, mycosis fungoides, fibrosarcoma
- Trauma
- Mass or nodular lesion with no evidence of bacteria or fungus of unknown cause (so called “idiopathic sterile granuloma”)
- Thickening of the skin of the nose of unknown cause (so called “idiopathic nasal hyperkeratosis”)

RISK FACTORS

- Adult cats—may be inapparent carriers of types of fungus (known as “dermatophytes”) that live on the skin, hair, or nails
- Rooting behavior—skin infection of the nose, characterized by the presence of pus (nasal pyoderma), superficial fungal infection (dermatophytosis)
- Sun exposure—inflammation of the skin in the lightly pigmented skin of the nose secondary to exposure to sunlight (nasal solar dermatitis) and autoimmune diseases (systemic lupus erythematosus, discoid lupus erythematosus, pemphigus erythematosus)
- Poorly pigmented nose—inflammation of the skin in the lightly pigmented skin of the nose secondary to exposure to sunlight (nasal solar dermatitis), skin cancer (squamous cell carcinoma)
- Large, rapidly growing breeds over supplemented with calcium or fed high-cereal diet—zinc-responsive skin disorder (zinc-responsive dermatosis)
- Decreased ability to produce a normal immune response (known as “immunosuppression”)—demodectic mange (demodicosis), skin infection characterized by the presence of pus (pyoderma), superficial fungal infection (dermatophytosis)



TREATMENT

HEALTH CARE

- Outpatient, except cases with systemic lupus erythematosus in which several organs are involved or with tumors requiring surgical removal and/or radiation therapy
- Reduce exposure to sunlight—inflammation of the skin in the lightly pigmented skin of the nose secondary to exposure to sunlight (nasal solar dermatitis); autoimmune diseases (systemic lupus erythematosus, discoid lupus erythematosus, pemphigus erythematosus), and skin cancer (squamous cell carcinoma)
- Discourage rooting behavior—skin infection of the nose, characterized by the presence of pus (nasal pyoderma), superficial fungal infection (dermatophytosis)
- Warm-water soaks—aid removal of discharge and dried discharge on the surface of the skin (crusts)
- Replace plastic or rubber dish and avoid contact with topical drug or other agent causing hypersensitivity reaction

ACTIVITY

- Limit sun exposure

SURGERY

- Surgical biopsy may be necessary for diagnosis
- Surgical removal may be necessary for treatment of tumors or cancer and for early localized fungal lesions
- Surgical removal of the mass or nodular lesion of unknown cause (idiopathic sterile granuloma), when feasible



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a

particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Depend on underlying cause; treatment should be directed at specific disease
- Fungal infections—medications to treat fungal infections administered by mouth (known as “systemic antifungals”): griseofulvin, ketoconazole, itraconazole (drug of choice in the cat); medication to treat fungal infections applied to the skin directly (known as “topical treatment”): enilconazole for aspergillosis
- Inflammation of the skin in the lightly pigmented skin of the nose secondary to exposure to sunlight (nasal solar dermatitis)—steroids applied to the nose directly (topical steroids); antibiotics for secondary infection; sunscreens; tattoo skin that is light or has lost pigment
- Mass or nodular lesion with no evidence of bacteria or fungus of unknown cause (idiopathic sterile granuloma)—medications to decrease the immune response (immunosuppressive therapy) with steroids and possibly with other medications (such as azathioprine, cyclosporine, tetracycline, and niacinamide)
- Systemic lupus erythematosus (an autoimmune disease that affects the skin and other organs of the body)—medications to decrease the immune response (immunosuppressive therapy) with steroids and possibly with other medications (such as azathioprine [dogs], chlorambucil, or gold salts [cats])
- Vitiligo or loss of pigment in the hair and/or skin of the nose (nasal depigmentation)—no treatment
- Tumors or cancer—surgical removal; chemotherapy; radiation therapy
- Thickening of the skin of the nose of unknown cause (idiopathic nasal hyperkeratosis)—antibiotic-steroid cream applied to the skin of the nose for furrows or slits (known as “fissures”) of the thickened skin; skin moistening product applied to the skin directly (known as a “topical humectant”), such as (KeraSolv® Gel from DVM Pharmaceuticals); topical tacrolimus (Protopic®)



FOLLOW-UP CARE

PATIENT MONITORING

- Varies with specific disease and treatment prescribed

PREVENTIONS AND AVOIDANCE

- Limit sun exposure (for cases with inflammation of the skin in the lightly pigmented skin of the nose secondary to exposure to sunlight [nasal solar dermatitis] or autoimmune diseases)

POSSIBLE COMPLICATIONS

- Scarring with deep infections or overly vigorous cleaning

EXPECTED COURSE AND PROGNOSIS

- Vary with specific disease



KEY POINTS

- Conditions characterized by abnormalities of the skin on the nose, involving either the haired portion (bridge of the nose) or non-haired portion (known as the “nasal planum”)
- Limit sun exposure (for cases with inflammation of the skin in the lightly pigmented skin of the nose secondary to exposure to sunlight [nasal solar dermatitis] or autoimmune diseases)
- Discourage rooting behavior
- Replace plastic or rubber food dish and avoid contact with topical drug or other agent causing hypersensitivity reaction

PAPULONODULAR DERMATOSES

(SKIN DISORDERS CHARACTERIZED BY THE PRESENCE OF BUMPS OR SMALL MASSES)



BASICS

OVERVIEW

- “Papulonodular” refers to the presence of papules and nodules; “dermatosis” (plural, “dermatoses”) is the medical term for any skin abnormality or disorder
- “Papules” are small, raised skin lesions of the skin; “nodules” are small, solid masses of the skin

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on underlying cause
- Hair loss (known as “alopecia”)
- Reddened skin (known as “erythema”)
- Small, raised skin lesions (papules)
- Small, solid masses of the skin (nodules)
- Small, raised skin lesions containing pus (known as “pustules”)
- Itchiness (known as “pruritus”)
- Skin lesions may be localized or generalized

CAUSES

- Bacterial folliculitis (superficial and deep bacterial infections of the hair follicles)
- Dermatophytosis (a fungal infection affecting the skin, hair, and/or nails)
- Sebaceous adenitis (inflammation of the sebaceous glands, the glands that produce oils in the hair coat)
- Sterile eosinophilic pustulosis (skin disorder characterized by the presence of eosinophils in sterile pustules; “eosinophils” are a type of white-blood cell; they are involved in allergic responses by the body and are active in fighting larvae of parasites)
- Canine and feline acne
- Kerions (raised nodular lesions that develop from fungal infections of the skin, which frequently ooze)
- Demodectic mange (known as “demodicosis”)
- Rhabditic dermatitis (skin inflammation caused by parasitic infestation [*Pelodera strongyloides*] in the skin)
- Actinic or sun/solar-related skin conditions
- Cutaneous histiocytosis (condition characterized by the proliferation of histiocytes, a type of scavenging cell, in the area of blood vessels in the skin)
- Cancer

RISK FACTORS

- Inflammation/infection of the hair follicles (folliculitis), fungal infection of the skin, hair, and/or nails (dermatophytosis), and demodectic mange (demodicosis)—any disease or medication that causes a decrease in the ability to produce a normal immune response (known as “immunocompromise”) increases likelihood of these skin disorders
- Rhabditic dermatitis—may be associated with contact with decaying organic debris (such as straw or hay) containing the parasite, *Pelodera strongyloides*
- Actinic or sun/solar-related skin conditions—seen more frequently in outdoor, short-haired dogs living in areas with ample sunlight



TREATMENT

HEALTH CARE

- Most can be treated as outpatients
- Generalized demodectic mange (demodicosis) and secondary generalized bacterial infection (known as “sepsis”) require hospitalization
- Some cases with skin cancer may require inpatient treatment

ACTIVITY

- Alteration of activity usually not necessary

DIET

- Alteration of diet usually not necessary

SURGERY

- Skin biopsy may be necessary to determine diagnosis
- Surgical removal of skin tumors/cancer



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Bacterial Infection of the Hair Follicles (Bacterial Folliculitis)

- Superficial skin infection characterized by the presence of pus (known as “superficial pyoderma”)—appropriate antibiotics based on bacterial culture and sensitivity testing should be given for at least 3 to 4 weeks or 1 week beyond resolution of clinical signs
- Deep skin infection characterized by the presence of pus (known as “deep pyoderma”)—appropriate antibiotics based on bacterial culture and sensitivity testing should be given for at least 6 to 8 weeks or 2 weeks beyond resolution of clinical signs

Inflammation of the Sebaceous Glands, the Glands that Produce Oils in the Hair Coat (Sebaceous Adenitis)

- Apply a 50% to 75% mixture of propylene glycol and water once daily as a spray to affected areas or apply baby oil weekly, as directed by your pet’s veterinarian
- Essential fatty acid dietary supplements and evening primrose oil, as directed by your pet’s veterinarian
- Cases that do not respond to treatment— isotretinoin; if response is seen, taper dosage as directed by your pet’s veterinarian (synthetic retinoids have become difficult to dispense due to very strict prescription procedures)
- Cyclosporine (medication to decrease the immune response) has been used
- Most cases do not respond to steroids

Canine and Feline Acne

- Underlying causes should be identified and treated accordingly
- May resolve without therapy in mild cases
- No underlying cause found—Stri-Dex® pads or benzoyl peroxide gels used daily or alternated daily, as directed by your pet’s veterinarian
- More severe cases—benzoyl peroxide has strong action to soften and loosen crusts and scales on the skin (known as “keratolytic action”), destroys bacteria or slows their growth and multiplication (known as an “antimicrobial property”), and flushes out the hair follicles; benzoyl peroxide shampoos and gels every 24 hours until lesions resolve, then as needed
- Mupirocin—antibiotic applied to the skin directly (known as a “topical antibiotic”); apply every 24 hours or alternate with the benzoyl peroxide products; should not be used in cats with deep lesions
- Cases that do not respond to treatment—antibiotics administered by mouth or injection (known as “systemic antibiotics”)
- Recurrent or very deep infection (furunculosis)—systemic antibiotics (administered by mouth or injection) and warm water soaks; “furunculosis” is a very deep bacterial infection leading to rupturing of the hair follicles
- Cases that are very resistant to treatment—tretinoin or isotretinoin applied to the skin directly (topical therapy); the synthetic retinoids intended to be administered by mouth (oral administration) have become difficult to dispense due to very strict prescription procedures
- Cats can be sensitive to the irritant effects of benzoyl peroxide

Rhabditic Dermatitis (skin inflammation caused by parasitic infestation [Pelodera strongyloides] in the skin)

- Remove and destroy bedding (adult parasites live in decaying organic material, such as straw)
- Wash kennels, beds, and cages and treat with a premise insecticide or flea spray, as directed by your pet’s veterinarian; always read labels on insecticides and follow instructions carefully
- Bathe affected animal and remove dried discharges on the surface of the skin lesions (known as “crusts”)
- Parasitocidal dip—at least 2 times at weekly intervals
- Severe infection—antibiotics may be necessary

Actinic or Sun/Solar-Related Skin Conditions

- Sunlight—avoid between 10 a.m. and 4 p.m.; apply sunscreen with SPF rating of at least 15 every 12 hours
- Severe inflammation—steroids applied directly to the affected skin (topical therapy) or administered by mouth may provide comfort; topical, 1%–2.5% hydrocortisone usually sufficient; systemic, prednisone by mouth
- Secondary infection—antibiotics may be necessary
- Squamous cell carcinoma (type of skin cancer)—prognosis is guarded to poor, depending on the stage of disease; therapy includes synthetic retinoids, heating of the tissues using radiofrequency waves (known as “radiofrequency hyperthermia”), freezing of the tissues (known as “cryosurgery”), use of a light sensitive drug, which is activated by certain light waves (known as “photochemotherapy”), radiation therapy, and surgical removal

Sterile Nodular Dermatoses

- Cyclosporine (medication to decrease the immune response)—no food 2 hours before or after dosing; taper according to response as directed by your pet’s veterinarian
- Tetracycline (an antibiotic) and niacinamide combinations
- Steroids at doses to decrease the immune response; taper according to response as directed by your pet’s veterinarian

- Chemotherapeutic drugs (chlorambucil or azathioprine)

Other Papulonodular Dermatoses

- Dermatophytosis—antifungal medications; may be administered by mouth or applied to the skin directly (topical therapy)
- Sterile eosinophilic pustulosis—steroids, prednisolone or prednisone
- Other papulonodular dermatoses treated based on underlying cause



FOLLOW-UP CARE

PATIENT MONITORING

- Blood work (complete blood count [CBC] and serum chemistry screen) and urinalysis—get baseline results, then repeat tests every 3 months for patients receiving cyclosporine
- Blood work (complete blood count [CBC] and serum chemistry screen) and urinalysis—monitor monthly for 4 to 6 months for patients receiving synthetic retinoid therapy
- Tear production—monitor monthly (perform Schirmer tear test) for 4 to 6 months, then every 6 months in patients receiving synthetic retinoid therapy
- Skin scrapings—monitor therapy in patients with demodectic mange (demodicosis)
- Repeat fungal (dermatophyte) cultures—monitor therapy in patients with fungal infection affecting the skin, hair, and/or nails (dermatophytosis)
- Monitor progress of resolution of lesions

PREVENTIONS AND AVOIDANCE

- Depend on underlying cause

POSSIBLE COMPLICATIONS

- Actinic or sun/solar skin conditions may progress to squamous cell carcinoma (type of skin cancer)

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause



KEY POINTS

- Fungal infection affecting the skin, hair, and/or nails (dermatophytosis)—contagious to humans in 30% to 50% of cases of *Microsporum canis*; if a family member develops skin lesions, seek medical attention

SEX HORMONE-RESPONSIVE SKIN DISORDERS (DERMATOSES)



BASICS

OVERVIEW

- “Dermatosis” (plural, dermatoses) is the medical term for any skin disorder
- Sex hormone-responsive skin disorders (dermatoses) are uncommon conditions characterized by hair loss (known as “alopecia”), suspected to result from an imbalance of sex hormones; often defined on the basis of response to sex hormone therapy
- “Estrogen,” “progesterone,” and “estradiol” are female hormones; “testosterone” and “androgen” are male hormones
- An “intact” animal is one that has its reproductive organs; an “intact female” has her ovaries and uterus and an “intact male” has his testicles
- A “neutered” animal has had its reproductive organs surgically removed; females commonly are identified as “spayed,” but may be identified as “neutered;” males may be identified as “castrated” or “neutered”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Estrogen-responsive skin disorders—dachshunds, boxers
- Condition characterized by the presence of fluid-filled sacs or cysts in the ovaries (known as “cystic ovaries”)—English bulldogs may be more susceptible than other breeds
- Increased levels of estrogen (known as “hyperestrogenism”) in male dogs due to testicular cancer—boxers, Shetland sheepdogs, Weimaraners, German shepherd dogs, Cairn terriers, Pekingese, and collies
- Testosterone-responsive skin disorders—Afghan hounds
- Castration-responsive skin disorders—chow chows, Samoyeds, keeshonden, Pomeranians, Siberian huskies, Alaskan malamutes, and miniature poodles
- Adrenal sex-hormone imbalance—Pomeranians

Mean Age and Range

- Estrogen-responsive skin disorders—primarily young adults
- Increased levels of estrogen (hyperestrogenism) in females—generally, middle-aged and old, intact female dogs
- Feminizing signs in a male dog of unknown cause (so called “idiopathic male feminizing syndrome”)—intact, middle-aged male dogs
- Testosterone-responsive skin disorders—old, castrated male dogs
- Castration-responsive skin disorders—onset of signs, 1 to 4 years of age or older
- Adrenal sex-hormone imbalance—onset of signs, 1 to 5 years of age

Predominant Sex

- Estrogen-responsive skin disorders—females
- Increased levels of estrogen (hyperestrogenism)—intact female dogs; male dogs due to testicular cancer producing excessive levels of estrogen
- Feminizing signs in a male dog of unknown cause (idiopathic male feminizing syndrome)—intact male dogs
- Testosterone-responsive skin disorders—castrated male dogs
- Castration-responsive skin disorders—intact male dogs with normal testicles
- Adrenal sex-hormone imbalance—males and females, intact or neutered

SIGNS/OBSERVED CHANGES in the ANIMAL

- Hair loss (alopecia)—localized hair loss (alopecia) more common than generalized; initially involves the skin between the anus and the external genitalia (known as the “perineum”), the under side of the chest and abdomen (known as the “ventrum”), thighs, and neck; later involves the back and flank; flank alopecia may be the first or only sign in some patients with increased levels of estrogen (hyperestrogenism) and may be seasonal in some spayed females
- Fur or hair coat—may be soft or dry and brittle
- Nipples, mammary glands, vulva (external genitalia of the female), prepuce, testicles, ovaries, and prostate—often abnormal

- Secondary excessively oily or dry scaling of the skin (known as “seborrhea”); itchiness (known as “pruritus”); skin infection characterized by the presence of pus (known as “pyoderma”); hair follicles filled with oil and skin cells (known as “comedones”); inflammation of the outer ear, characterized by an oily discharge (known as “ceruminous otitis externa”); and darkened skin (known as “hyperpigmentation”)—variable
- Increase in the number of cells in the tail glands (known as “tail gland hyperplasia”) and increase in the number of cells in the perianal gland (known as “perianal gland hyperplasia”) with localized change in color of the skin due to deposits of melanin (known as “macular melanosis”)—dogs with testicular tumors
- Urinary incontinence—estrogen- and testosterone-responsive conditions

CAUSES

Estrogen-Responsive Skin Disorders—Females

- Possible deficiency or imbalance of the female hormone, estrogen; serum estradiol concentrations may be normal
- Inadequate production of adrenal sex hormones
- Skin defect in the sex hormone-receptor/metabolism system
- Rare in dogs
- Extremely rare in cats
- Susceptible breeds—dachshunds and boxers
- Primarily seen in young adults
- May occur after surgical removal of the ovaries and uterus (known as a “spay” or “ovariohysterectomy”) in non-cycling, intact females
- Occasionally seen during false pregnancy or pseudopregnancy
- Variant—cyclical flank hair loss (alopecia) and darkened skin (hyperpigmentation); noted in Airedale terriers, boxers, and English bulldogs; may worsen in winter

Skin Disorders due to Increased Levels of Estrogen (Hyperestrogenism)—Females

- Estrogen excess or imbalance owing to a condition characterized by the presence of fluid-filled sacs or cysts in the ovaries (cystic ovaries), ovarian tumors (rare), or excess/overdose of estrogen-containing medications
- Abnormal peripheral conversion of sex hormones
- Production of sex hormones in an unexpected location in the body
- Animals with normal serum estrogen concentrations may have an increased number of estrogen receptors in the skin
- Rare in dogs
- Extremely rare in cats
- English bulldogs may be more susceptible to a condition characterized by the presence of fluid-filled sacs or cysts in the ovaries (cystic ovaries) than other breeds
- Generally, middle-aged and old, intact female dogs

Skin Disorders due to Increased Levels of Estrogen (Hyperestrogenism)—Male Dogs with Testicular Tumors

- Estrogen excess (or rarely increased levels of the female hormone, progesterone [known as “hyperprogesteronism”]) due to a tumor in the testicles, such as Sertoli cell tumor (most common), seminoma, or interstitial cell tumor (rarely)
- Lack of normal descent of one or both testicles into the scrotum, resulting in the testicle(s) being located in the abdomen or inguinal canal (known as “cryptorchidism”) increases the likelihood that affected animals will develop testicular tumors
- Intact males; usually middle-aged or older
- Susceptible breeds—boxers, Shetland sheepdogs, Weimaraners, German shepherd dogs, Cairn terriers, Pekingese, and collies
- Associated with male pseudohermaphroditism in miniature schnauzers; “pseudohermaphroditism” is a condition where the animal has either ovaries or testicles, but has uncertain (ambiguous) external genitalia

Skin Disorder due to Increased Levels of Androgen (known as “Hyperandrogenism”) Associated with Testicular Tumors

- Androgen-producing testicular tumors (especially interstitial cell tumors) in intact male dogs

Skin Disorder and Feminizing Signs in a Male Dog for Unknown Cause (Idiopathic Male Feminizing Syndrome)

- Undetermined cause
- Serum sex hormone concentrations normal
- Blockage of androgen receptors in the skin may prevent attachment of testosterone
- Intact, middle-aged male dogs

Testosterone-Responsive Skin Disorders—Males

- Rare
- Old, castrated male dogs
- Afghan hounds may be more susceptible than other breeds

- Extremely rare in cats
- Suspected low levels of androgen (known as “hypoandrogenism”) or a possible defect in the skin sex hormone–receptor system

Castration-Responsive Skin Disorders

- Intact males with normal testicles
- Hormone levels (estradiol, testosterone, and progesterone)—variably high, low, or normal
- Onset of signs, 1 to 4 years of age or older
- Susceptible breeds—chow chows, Samoyeds, keeshonden, Pomeranians, Siberian huskies, Alaskan malamutes, and miniature poodles

Adrenal Sex-Hormone Imbalance

- Adrenal enzyme (21-hydroxylase) deficiency, resulting in excessive adrenal gland secretion of androgen or progesterone
- Males and females, intact or neutered
- Onset of signs, 1 to 5 years of age
- Pomeranians may be more susceptible than other breeds



TREATMENT

HEALTH CARE

- Depends on type of sex hormone-responsive skin disorder (dermatosis)
- Discontinue administration of estrogen-containing medications, as directed by your veterinarian, if excessive estrogen is the likely cause of the skin disorder

SURGERY

- Skin biopsy
- Surgical removal of testicles (neuter or castration) of animals with lack of normal descent of one or both testicles into the scrotum, resulting in the testicle(s) being located in the abdomen or inguinal canal (cryptorchidism); neuter when young
- Surgical removal of testicles (neuter or castration)—castration-responsive skin disorder (dermatosis) and testicular tumors
- Exploratory surgery (known as a “laparotomy”)—diagnosis and treatment (such as surgical removal of the ovaries and uterus [spay or ovariectomy] and surgical removal of testicles located in the abdomen [castration]) for ovarian cysts and tumors and abdominal testicular tumors



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

General Treatment

- Topical (applied to the skin directly) medication to treat seborrhea (known as “antiseborrheic therapy”)—conditions with associated keratinization defects and comedones (in which the hair follicles are filled with oils and skin cells)
- Antibiotics—to treat associated skin infections, characterized by the presence of pus (pyoderma)
- Prednisone—for itchiness (pruritus), if infections and bone-marrow suppression have been ruled out

Estrogen-Responsive Skin Disorder (Dermatosis)

- Spayed females—diethylstilbestrol (DES); if no response, try methyltestosterone or mibolerone until hair regrowth and then taper to maintenance
- Intact females—diethylstilbestrol (DES) until bloody vaginal discharge; then administer luteinizing hormone and finally follicle-stimulating hormone (treatment is designed for administration of hormones on specific days of the induced “heat” or “estrous” cycle, as administered or directed by your pet’s veterinarian; “luteinizing hormone” is a female hormone that stimulates the ovarian follicle to complete development and rupture to allow release of the egg and to produce progesterone; “follicle-stimulating hormone” is a hormone from the pituitary gland that stimulates development of eggs in the ovaries and production of estrogen)
- Alternative treatment—follicle-stimulating hormone until signs of “heat” or “estrus” appear

Testosterone-Responsive Skin Disorder (Males)/Some Estrogen-Responsive Skin Disorders (Female Dogs)

- Methyltestosterone—until response; may take 1 to 3 months; after hair regrowth is complete, administer 2 to 3 times/week for maintenance

- Repositol testosterone as needed to maintain normal hair coat

Skin Disorders due to Increased Levels of Estrogen (Hyperestrogenism)—Female Dogs

- Consider o,p'-DDD (Lysodren®) or l-deprenyl
- Alternative treatments—gonadotropin-releasing hormone (hormone that causes release of luteinizing hormone and follicle-stimulating hormone from the pituitary gland) or human chorionic gonadotropin (hormone produced early in pregnancy by the human embryo to maintain progesterone production by the “corpus luteum” or “yellow body” in the ovary; progesterone supports and maintains the pregnancy)
- Tamoxifen (drug that competes with estrogen for receptor sites)—may be useful

Other Conditions

- Castration-responsive hair loss (alopecia)—may respond to human chorionic gonadotropin (hormone produced early in pregnancy by the human embryo to maintain progesterone production by the “corpus luteum” or “yellow body” in the ovary; progesterone supports and maintains the pregnancy) or testosterone, if castration is not possible
- Adrenal 21-hydroxylase enzyme deficiency—o,p'-DDD (Lysodren®), if adrenal sex hormones are high



FOLLOW-UP CARE

PATIENT MONITORING

- Diethylstilbestrol (DES) treatment—complete blood count (CBC) to monitor for decreased production of blood cells by the bone marrow (known as “bone-marrow hypoplasia”) or lack of production of blood cells (known as “bone-marrow aplasia”) every 2 weeks for the first month; then every 3 to 6 months
- Testosterone treatment—blood work (serum biochemistry) with an emphasis on liver enzymes every 3 to 4 weeks for the first 3 months; then every 4 to 6 months
- Treatment with o,p'-DDD (Lysodren®)—blood work (electrolytes) and adrenocorticotrophic hormone (ACTH)-stimulation testing every 3 months

PREVENTIONS AND AVOIDANCE

- Animals with lack of normal descent of one or both testicles into the scrotum, resulting in the testicle(s) being located in the abdomen or inguinal canal (cryptorchidism)—do not breed

POSSIBLE COMPLICATIONS

- Estrogen treatment or excessive estrogen—decreased production of blood cells by the bone marrow (bone-marrow hypoplasia) or lack of production of blood cells (bone-marrow aplasia), which are uncommon; signs of “heat” or “estrus,” which is rare
- Methyltestosterone treatment—inflammation of the bile ducts and liver (known as “cholangiohepatitis”), which is rare; behavior changes (uncommon); excessive oily scaling of the skin (known as “seborrhea oleosa”)
- Treatment with o,p'-DDD (Lysodren®)—potential side effects (such as vomiting, diarrhea, collapse, and inadequate production of steroids by the adrenal glands secondary to medical treatment [known as “iatrogenic hypoadrenocorticism”])
- Tamoxifen—swelling of the vulva (female genitalia); discontinue until signs of “heat” or “estrus” are gone

EXPECTED COURSE AND PROGNOSIS

- Estrogen-responsive skin disorder (dermatosis)—regrowth of hair may take about 3 months and may be transient
- Female increased levels of estrogen (hyperestrogenism)—improvement should occur within 3 to 6 months after surgical removal of the ovaries and uterus (spay or ovariectomy)
- Estrogen- and androgen-secreting tumors—resolution of signs noted within 3 to 6 months after surgical removal of the ovaries and uterus (spay or ovariectomy) or the testicles (castration), respectively; lack of production of blood cells (bone-marrow aplasia) associated with hyperestrogenism usually does not respond to neutering, and the prognosis for recovery is grave; relapse after a positive response to castration may indicate spread of cancer (known as “metastasis”), and if confirmed, the prognosis is poor
- Castration-responsive skin disorder (dermatosis)—response noted 2 to 4 months after castration
- Testosterone therapy—may result in hair regrowth in 4 to 12 weeks
- Adrenal sex-hormone imbalance—response seen 4 to 12 weeks after treatment with o,p'-DDD (Lysodren®) to decrease the production of hormones by the adrenal glands



KEY POINTS

- Sex hormone-responsive skin disorders (dermatoses) are uncommon conditions characterized by hair loss (alopecia), suspected to result from an imbalance of sex hormones; often defined on the basis of response to sex hormone therapy

EROSIONS OR ULCERS OF THE SKIN (EROSIVE OR ULCERATIVE DERMATOSES)



BASICS

OVERVIEW

- Erosions are shallow defects in the skin, which only affect the skin's upper layers; erosions can be quite painful, but tend to heal quickly if protected (and the underlying cause is eliminated)
- Ulcers are deeper defects in the skin, where the surface layers are compromised completely; ulcers require careful wound care to prevent infection, and tend to heal slowly
- Erosive or ulcerative dermatoses are a group of dissimilar skin disorders, characterized by the presence of erosions or ulcers

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on cause
- Erosions or ulcers; they may be found anywhere on the body
- Hair loss (known as "alopecia")
- Single or multiple lesions; lesions may be inflamed (indicated by redness)
- May see lesions over pressure points (such as skin over bones)
- May have dried discharge on the surface of a skin lesion (known as a "crust") or may have moist discharge
- May have loss of pigment of skin and/or hair (known as "depigmentation")

CAUSES

- Wide variety of diseases may result in erosions or ulcers of the skin; common causes are burns, trauma, and skin infections; more complicated diseases, such as drug reactions, certain types of cancers, autoimmune diseases of the skin, and viruses also may cause erosions or ulcers that appear identical to burns or trauma—your pet's veterinarian may need to run a battery of tests (including blood work, cultures for different types of infections, and skin biopsies) to identify the cause and prescribe proper treatment
- In some cases, an underlying cause cannot be identified and the cause is "unknown," so called "idiopathic" disorder or disease
- Partial list of disorders that cause erosions or ulcers of the skin include the following:

Immune-Mediated Disorders

- Inflammation of blood vessels (known as "vasculitis")
- Canine juvenile cellulitis (puppy strangles)
- Toxic epidermal necrolysis (usually medication-induced)
- Feline indolent ulcer (rodent ulcer)
- Pemphigus, an autoimmune disorder in which the immune system attacks the skin

Infectious Disorders

- Skin infection characterized by the presence of pus (known as "pyoderma") caused by *Staphylococcus*
- Deep fungal or mycotic infections (such as sporotrichosis, cryptococcosis, histoplasmosis)
- Superficial fungal infections (*Malassezia* dermatitis, dermatophytosis)
- Actinomycetic bacteria (such as *Nocardia*, *Actinomyces*, *Streptomyces*)
- Feline cow pox
- Feline immunodeficiency virus (FIV) and feline leukemia virus (FeLV)-related disease

Parasitic Disorders

- Demodectic mange (demodicosis)
- Sarcoptic or notoedric mange
- Flea-bite allergy

Congenital/Hereditary Disorders

- Various skin disorders in which the skin is abnormal at birth (that is, a "congenital" abnormality) that may or may not be inherited

Metabolic Disorders

- Excessive production of steroids by the adrenal glands (known as "hyperadrenocorticism" or "Cushing's disease"), especially when complicated by secondary infections or calcium deposits in the skin (known as "calcinosis cutis")

Cancer

- Squamous cell carcinoma
- Mast cell tumors
- Lymphoma of the skin ("mycosis fungoides")

Nutritional Disorder

- Zinc-responsive dermatosis
- Generic dog-food dermatosis

Miscellaneous

- Thermal, electrical, solar, or chemical burns
- Frost bite
- Chemical irritants
- Venomous snake and insect bites

RISK FACTORS

- Depend on underlying cause



TREATMENT

HEALTH CARE

- Outpatient for most diseases
- Varies widely according to the cause
- Keeping eroded or ulcerated skin clean and protected are key to healing; if the cause is known, specific drug therapies may be prescribed
- Your veterinarian will tailor a management program that is best for your pet's individual case
- Hydrotherapy, which may be achieved with either a whirlpool bath or by spraying cool water under pressure against the ulcerated skin can be helpful in many cases; ask your pet's veterinarian first to be sure that hydrotherapy is appropriate for your pet's condition
- Avoid the temptation to apply "over-the-counter" creams and ointments to erosions and ulcers, without first checking with your veterinarian—some commonly used products (such as those containing neomycin) actually may delay healing in some cases; other products may contain types of alcohol or other ingredients that could cause pain upon application

DIET

- Supportive therapy with fluid and nutritional supplementation is indicated in cases with severe fluid and protein loss through the damaged skin
- Good quality diet

SURGERY

- Skin biopsy may be necessary for diagnosis



MEDICATIONS

- Vary widely according to cause



FOLLOW-UP CARE

PATIENT MONITORING

- Case-by-case basis, depending on the disease process, presence of generalized (systemic) disease(s), medications used, and potential side effects expected
- Follow-up care is important, especially for slowly healing ulcers; a veterinarian should check progress of the wound at least every-other-week to be sure that healing is proceeding properly, and that infection has not complicated the healing process

POSSIBLE COMPLICATIONS

- Depend on cause
- Some diseases are potentially life-threatening
- Some diseases are caused by agents that may be spread to people (known as having "zoonotic potential")
- Superinfections and drug side effects are possible in cases requiring medications to decrease the body's immune response (known as "immunosuppression")
- Some infectious diseases (such as nocardiosis, atypical mycobacteriosis) may be controlled, but not cured

EXPECTED COURSE AND PROGNOSIS

- Vary widely according to cause



KEY POINTS

- Wide variety of diseases may result in erosions or ulcers of the skin; common causes are burns, trauma, and skin infections; more complicated diseases, such as drug reactions, certain types of cancers, autoimmune diseases of the skin, and viruses also may cause erosions or ulcers that appear identical to burns or trauma—your pet's veterinarian may need to run a battery of tests (including blood work, cultures for different types of infections, and skin biopsies) to identify the cause and prescribe proper treatment
- Follow-up care is important, especially for slowly healing ulcers; a veterinarian should check progress of the wound at least every-other-week to be sure that healing is proceeding properly, and that infection has not complicated the healing process

VESICULOPUSTULAR DERMATOSES

(SKIN DISORDERS CHARACTERIZED BY BLISTERS AND/OR PUSTULES)



BASICS

OVERVIEW

- “Vesiculo-“ refers to vesicles; “pustular” refers to pustules; “dermatoses” is the plural of “dermatosis,” which is used to describe any skin abnormality or disorder
- Vesicle—blister; small, circumscribed elevation of the outer layer of the skin (known as the “epidermis”) filled with clear fluid
- Pustule—small, circumscribed elevation of the outer layer of the skin (epidermis) filled with pus

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Lupus erythematosus—collies, shelties, and German shepherd dogs may be more susceptible than other breeds
- Pemphigus erythematosus—collies and German shepherd dogs may be more susceptible than other breeds
- Pemphigus foliaceus—Akitas, chow chows, dachshunds, bearded collies, Newfoundlands, Doberman pinschers, and schipperkes may be more susceptible than other breeds
- Bullous pemphigoid—collies and Doberman pinschers may be more susceptible than other breeds
- Dermatomyositis—young collies and Shetland sheepdogs
- Subcorneal pustular dermatosis—schnauzers affected most frequently
- Linear IgA dermatosis—dachshunds exclusively

Mean Age and Range

- Dermatomyositis (fungal skin infection)—young animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on disease
- Hair loss (known as “alopecia”) and reddened skin (known as “erythema”)
- Presence of vesicles (blisters; small, circumscribed elevations of the outer layer of the skin filled with clear fluid)
- Presence of pustules (small, circumscribed elevations of the outer layer of the skin filled with pus)
- Loss of pigment of the skin and/or hair (known as “depigmentation”)

CAUSES

Vesicles

- Systemic lupus erythematosus (SLE; autoimmune disease in which body attacks its own skin and possibly other organs)
- Discoid lupus erythematosus (DLE; autoimmune disease involving the skin only, usually the face)
- Bullous pemphigoid (autoimmune disease with ulceration of the skin and/or moist tissues [known as “mucous membranes”] of the body)
- Pemphigus vulgaris (severe autoimmune disease with ulceration of the mouth, junction between the moist tissues [mucous membranes] and skin, and skin)
- Dermatomyositis (inflammatory disorder that affects the skin and muscles) in collies and Shetland sheepdogs

Pustules

- Skin infection involving the surface or top of the skin (known as a “superficial skin infection”) characterized by the presence of pus (known as “pyoderma”)—bacterial skin infection involving the areas of the body with sparse hair coat (known as “impetigo”), superficial spreading pyoderma, superficial bacterial infection/inflammation of the hair follicles (known as “bacterial folliculitis”), acne
- Pemphigus complex—pemphigus foliaceus, pemphigus erythematosus, pemphigus vegetans (autoimmune skin diseases)
- Subcorneal pustular dermatosis (skin disease of unknown cause characterized by the presence of pustules)
- Dermatomyositis (fungal skin infection)
- Sterile eosinophilic pustulosis (skin disorder characterized by the presence of eosinophils in the pustules; “eosinophils” are a type of white-blood cell; they are involved in allergic responses by the body and are active in fighting larvae of parasites)
- Linear immunoglobulin A (IgA) dermatosis (a skin disorder seen only in dachshunds in which sterile pustules are located just below the surface of the skin; immunoglobulin A [IgA] is present in the lowest layer of the epidermis [known as the “basement membrane”]; “immunoglobulins” are proteins produced by the cells of the immune system; they include the antibodies; they are categorized into classes, including immunoglobulin A [IgA])

RISK FACTORS

- Drug exposure—autoimmune diseases (systemic lupus erythematosus [SLE] and bullous pemphigoid)
- Pyoderma (skin infection characterized by presence of pus) usually are secondary to a predisposing factor (such as demodectic mange, inadequate levels of thyroid hormone [known as “hypothyroidism”], allergy, or steroid administration)
- Sunlight—pemphigus erythematosus, bullous pemphigoid, systemic lupus erythematosus (SLE), discoid lupus erythematosus (DLE), and dermatomyositis (inflammatory disorder that affects the skin and muscles in collies and Shetland sheepdogs)



TREATMENT

HEALTH CARE

- Periodic bathing with an antimicrobial shampoo—helps remove surface debris and control secondary bacterial infections
- Usually treated as an outpatient
- Systemic lupus erythematosus (SLE), pemphigus vulgaris, and bullous pemphigoid may be life-threatening and require inpatient intensive care

SURGERY

- Surgical biopsy may be necessary to determine diagnosis



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Pemphigus Complex/Bullous Pemphigoid (Autoimmune Diseases)

- Chemotherapeutic drugs—azathioprine or chlorambucil; used to decrease the immune response
- Tetracycline and niacinamide combination
- Cyclosporine (Neoral®)—used to decrease the immune response

Subcorneal Pustular Dermatitis

- Dapsone—until remission (usually 1 to 4 weeks); then tapered as directed by your pet's veterinarian
- Sulfasalazine (Azulfidine®)—until remission; then as needed as directed by your pet's veterinarian

Linear Immunoglobulin A (IgA) Dermatitis

- Prednisolone—until remission; then taper as directed by your pet's veterinarian
- Dapsone—until remission; then taper and give as needed (as directed by your pet's veterinarian); individual patients may respond to one drug and not the other

Sterile Eosinophilic Pustulosis

- Prednisolone—until remission (usually 5 to 10 days); then as needed to prevent relapses (as directed by your pet's veterinarian); usually long-term treatment is required



FOLLOW-UP CARE

PATIENT MONITORING

- Dapsone—monitor blood work (complete blood count [CBC], platelet count, and liver enzymes) every 2 weeks initially and if any clinical side effects develop
- Long-term sulfasalazine therapy—monitor tear production
- Treatment to decrease the immune response (known as “immunosuppressive therapy”)—monitor every 1 to 2 weeks initially; then every 3 to 4 months during maintenance therapy

POSSIBLE COMPLICATIONS

- Depend on underlying cause

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause
- Systemic lupus erythematosus (SLE), pemphigus vulgaris, and bullous pemphigoid may be life-threatening

DIABETES INSIPIDUS (“WATER DIABETES”)



BASICS

OVERVIEW

- Diabetes insipidus is a disorder of water metabolism characterized by excessive urination (polyuria) and excessive thirst (polydipsia); the urine is very watery with low levels of dissolved substances (such as salt)—the urine is described as having a low specific gravity or osmolality on the urinalysis; so-called “insipid urine” or “tasteless urine”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog and cat

Mean Age and Range

- Congenital (present at birth) forms—less than 1 year of age
- Acquired (condition develops at some time after birth) forms (such as caused by cancer or trauma or for unknown reasons [known as “idiopathic”])—any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Excessive urination (polyuria)
- Excessive thirst (polydipsia)
- Inability to control urination (incontinence)—occasional

CAUSES

Two general types of diabetes insipidus have been identified, based on the location of the water metabolism defect. One type is known as “central diabetes insipidus” in which the pituitary gland does not produce enough antidiuretic hormone (ADH). Antidiuretic hormone normally is involved in regulating water balance in the body. Antidiuretic hormone acts on the kidney to increase the amount of water reabsorbed so that normal levels of fluid are present in the blood. If the body has too much fluid present, the pituitary does not secrete ADH so the kidney does not reabsorb as much water, and more water enters the urine and is eliminated from the body. The other type is called “nephrogenic diabetes insipidus.” Nephrogenic refers to the kidney itself. In this type of diabetes insipidus, the kidney does not respond to the ADH, such that the kidney does not reabsorb water and excessive urine is produced.

Inadequate Secretion of ADH by the Pituitary Gland (Central Diabetes Insipidus)

- Congenital defect
- Unknown problem in the pituitary gland (idiopathic)
- Trauma to the brain/pituitary gland
- Cancer

Kidney Insensitivity to ADH (Nephrogenic Diabetes Insipidus)

- Congenital
- Secondary to drugs (such as lithium, demeclocycline, and methoxyflurane)
- Secondary to hormone/endocrine and metabolic disorders (such as excessive production of steroids by the adrenal glands [hyperadrenocorticism], low potassium levels in the blood [hypokalemia], accumulation of pus in the uterus [pyometra], and high calcium levels in the blood [hypercalcemia])
- Secondary to kidney disease or infection (such as inflammation and/or infection of the kidney [known as “pyelonephritis”], chronic kidney failure, kidney disease related to accumulation of pus in the uterus [pyometra])



TREATMENT

HEALTH CARE

- Patients should be hospitalized for the modified water deprivation test; the ADH trial uses synthetic antidiuretic hormone administered to the pet to determine if the kidneys will respond—it often is performed as an outpatient procedure

ACTIVITY

- Not restricted

DIET

- Normal, with free access to water at all times



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Central diabetes insipidus—desmopressin acetate or DDAVP is a synthetic antidiuretic hormone; intranasal and oral preparations are available; your pet's veterinarian will determine the type to be used and the route of administration
- Nephrogenic diabetes insipidus—hydrochlorothiazide
- Chlorpropamide (Diabinese®) may reduce excessive urination and thirst in cases of central diabetes insipidus



FOLLOW-UP CARE

PATIENT MONITORING

- Treatment is adjusted according to the patient's signs; the ideal dosage and frequency of DDAVP administration is based on water intake
- Laboratory tests, such as packed cell volume (PCV), total solids, and serum sodium concentration to detect dehydration (could indicate inadequate DDAVP replacement)

PREVENTIONS AND AVOIDANCE

- Avoid circumstances that might increase water loss markedly
- Ensure free access to water at all times to prevent dehydration

POSSIBLE COMPLICATIONS

- Complications of primary disease (for example, pituitary tumor)

EXPECTED COURSE AND PROGNOSIS

- The condition is usually permanent, except in rare patients in which the condition was trauma induced
- Prognosis is generally good, depending on the underlying disorder
- Without treatment, dehydration can lead to stupor, coma, and death



KEY POINTS

- Diabetes insipidus is a disorder of water metabolism characterized by excessive urination (polyuria) and excessive thirst (polydipsia); the urine is very watery with low levels of dissolved substances (such as salt)
- Ensure free access to water at all times to prevent dehydration

UNCOMPLICATED DIABETES MELLITUS IN CATS



BASICS

OVERVIEW

- Disorder of carbohydrate, fat, and protein metabolism caused by absolute or relative insulin deficiency
- Type II (non-insulin-dependent diabetes mellitus) is characterized by inadequate or delayed insulin secretion relative to the needs of the patient; many of these patients live without the need for insulin injections (exogenous insulin) and are less prone to ketoacidosis (acidic condition of the blood caused by the presence of ketone bodies); most common form in cats
- “Uncomplicated” diabetes mellitus is a designation that indicates the cat has diabetes mellitus, but does not have secondary problems (such as ketoacidosis, vomiting, or diarrhea) that makes the cat “more sick” and requires more aggressive treatment

GENETICS

- Genetic susceptibility

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Breed Predilections

- None

Mean Age and Range

- 75% are 8 to 13 years of age; range, 1 to 19 years of age

Predominant Sex

- Male

SIGNS/OBSERVED CHANGES in the ANIMAL

- Early signs—obesity with recent weight loss is typical
- Muscle wasting along the back and an oily coat with dandruff are common
- Enlarged liver (known as “hepatomegaly”); yellowish discoloration to skin and moist tissues (mucous membranes) of the body (icterus or jaundice) more common
- Less common findings—a plantigrade stance (in which the lower part of the rear legs is dropped toward the ground so it looks like the cat is standing or walking on its lower leg and paw or is in a somewhat crouched position; caused by a nervous system disorder related to the diabetes, known as “diabetic neuropathy”)
- Later signs—excessive urination (polyuria) and excessive thirst (polydipsia), excessive appetite (polyphagia), and weight loss, loss of appetite (anorexia), sluggishness (lethargy), depression, and vomiting

CAUSES

- Genetic susceptibility
- Amyloid (insoluble proteins deposited outside of the cells in various tissues and organs, compromising their normal function)
- Inflammation of the pancreas (pancreatitis); the pancreas is the organ that normally produces and releases insulin
- Predisposing diseases (such as hyperadrenocorticism or Cushing’s disease [where the adrenal glands produce excessive amounts of steroids] and acromegaly [where the body produces excessive amounts of somatotropin, a growth hormone, leading to enlargement of the head, limbs, and organs])
- Drugs (such as steroids and progestogens)

RISK FACTORS

- Obesity for type II diabetes mellitus



TREATMENT

HEALTH CARE

- Cats that have compensated for their diabetes (that is, the cat appears to be “healthy” and is alert, eating and drinking, with no signs of vomiting) can be managed as outpatients

ACTIVITY

- Strenuous activity may lower insulin requirement; a consistent amount of activity each day is helpful

DIET

- Avoid soft, moist foods because they cause severe increases in blood glucose (hyperglycemia) following the meal (known as “postprandial elevations in blood glucose”)
- Thin cats or non-obese cats—feed a high-protein, low-carbohydrate diet that the pet will eat reliably; keep daily caloric intake constant; avoid reduced-calorie diet; starvation worsens ketoacidosis (acidic condition of the blood caused by the presence of ketone bodies) and may lead to poor immune function
- Obese cats—gradual weight reduction improves insulin sensitivity and reverses diabetes in some cats with type II diabetes mellitus; feed a high-protein, low-carbohydrate diet
- Special considerations for feline diabetics: cats evolved as obligate carnivores (they must have meat in their diet) with metabolic pathways adapted for efficient utilization of protein, but not well suited for large-carbohydrate loads; preliminary studies suggest that low-carbohydrate canned diets may lower insulin requirements in diabetic cats; most dry pet food is manufactured with a higher carbohydrate content than the equivalent wet form, and simply switching from dry to wet food may reduce percentage of body fat and improve control of blood glucose levels (known as “glycemic control”) in obese, diabetic cats; any change in diet should be under the supervision of your pet’s veterinarian; monitor closely for change in insulin requirement following any adjustment in diet

SURGERY

- Intact females should be spayed (ovariohysterectomy) when stable; progesterone (a female hormone) secretion makes management of diabetes mellitus difficult



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Insulin—treatment of choice for most cats
- Pork lente insulin (Vetsulin™, U-40)—intermediate duration of effect; given subcutaneously (under the skin)
- Protamine zinc insulin (PZI, U-40)—long-acting insulin; given subcutaneously
- Glargine insulin (Lantus®, U-100)—human recombinant insulin; gives best results when combined with a high-protein, low-carbohydrate, diet—remission rates of 70% to 100% reported with this combination
- Species of origin of the insulin may affect how the drug is absorbed and used in the body (known as “pharmacokinetics”); beef, beef/pork, human recombinant insulin are options; beef preferred for cats
- Oral drugs to decrease blood glucose (known as “hypoglycemic agents”)—glipizide is useful with dietary therapy in cats with type II diabetes mellitus; the cat should have uncomplicated diabetes mellitus (“uncomplicated” diabetes mellitus is a designation that indicates the cat does not have secondary problems [such as ketoacidosis, vomiting, or diarrhea] that makes the cat “more sick” and requires more aggressive treatment) and no history of ketoacidosis (acidic condition of the blood caused by the presence of ketone bodies)
- Acarbose



FOLLOW-UP CARE

PATIENT MONITORING

- Glucose curve—not helpful in cats
- Urinary glucose monitoring—urine is tested for glucose and ketones before the meal and insulin injection; to use this as a regulatory method, the pet must be allowed to have trace to 1/4% glucose in the urine (glucosuria) to avoid the development of too low a level of glucose in the blood (hypoglycemia)
- Fructosamine (a particular protein found in blood used to monitor control of blood glucose levels [glycemic control])—maintain at less than 400 micromol/L; recheck every four weeks during initial regulation of insulin treatment and then every three months
- Clinical signs—owner can assess degree of excessive urination (polyuria) and excessive thirst (polydipsia), appetite, and body weight; if these are normal, the disease is well regulated or controlled

PREVENTIONS AND AVOIDANCE

- Prevent or correct obesity
- Avoid unnecessary treatment with steroids or megestrol acetate

POSSIBLE COMPLICATIONS

- Seizure or coma with insulin overdose
- Low red blood-cell count (anemia) and the presence of hemoglobin in the blood (hemoglobinemia) with severely low levels of phosphorus in the blood (hypophosphatemia), which can occur after initial insulin therapy
- Nerve damage related to diabetes mellitus (diabetic neuropathy)

EXPECTED COURSE AND PROGNOSIS

- Some cats recover, but may relapse at a later time

- Prognosis with treatment is good; most animals have normal life spans



KEY POINTS

- Daily feeding and medication schedules are necessary to maintain blood glucose in normal range
- If the cat develops signs of weakness, sluggishness, or seizures (possible signs of low blood sugar [hypoglycemia]) call your pet's veterinarian immediately
- Keep a chart of pertinent information about the pet, such as urine dipstick results, daily insulin dose, and weekly body weight

UNCOMPLICATED DIABETES MELLITUS IN DOGS



BASICS

OVERVIEW

- Increased levels of glucose (sugar) in the blood (known as “hyperglycemia”) when the dog has been fasted, combined with the presence of glucose (sugar) in the urine (known as “glucosuria”)
- Disorder of carbohydrate, fat, and protein metabolism caused by an absolute or relative insulin deficiency
- The pancreas is an organ of the body, located near the upper small intestine; the pancreas produces insulin to regulate blood sugar
- Type I diabetes mellitus is characterized by destruction of insulin-secreting pancreatic β -cells by the body’s own immune system (that is, it is an autoimmune destruction of the β -cells) and results in a dependence on insulin treatment (also known as “insulin-dependent diabetes mellitus” or “IDDM”)
- Type II diabetes mellitus is characterized by a relative insulin deficiency (either inadequate insulin secretion or lack of response of the body to insulin [known as “insulin resistance”]); type II may result in “insulin-dependent diabetes mellitus” or “IDDM;” “non–insulin dependent diabetes mellitus” or “NIDDM;” or both through the course of the disease
- Insulin-dependent diabetes mellitus or IDDM patients are prone to developing diabetic ketoacidosis (condition in which levels of acid are increased in the blood due to the presence of ketone bodies secondary to diabetes)
- Non-insulin dependent diabetes mellitus or NIDDM patients may respond to treatment with medications administered by mouth to decrease blood glucose (known as “oral hypoglycemic agents”)
- “Uncomplicated” diabetes mellitus is a designation that indicates the dog has diabetes mellitus, but does not have secondary problems (such as ketoacidosis, vomiting, or diarrhea) that makes the dog “more sick” and requires more aggressive treatment
- Diabetes mellitus also is known as “sugar diabetes”

GENETICS

- Familial (runs in certain families or lines of animals) associations in some breeds of dog

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- The keeshond, puli, miniature pinscher, and Cairn terrier are at higher risk than other breeds
- The poodle, dachshund, miniature schnauzer, and beagle possibly are at higher risk than other breeds

Mean Age and Range

- Mean, approximately 8 years of age; range, 4 to 14 years (excluding rare juvenile form)

Predominant Sex

- Female

SIGNS/OBSERVED CHANGES in the ANIMAL

- Early signs—increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”), increased appetite (known as “polyphagia”), and weight loss
- Later signs—lack of appetite (known as “anorexia”), sluggishness (lethargy), depression, and vomiting
- Enlargement of the liver (known as “hepatomegaly”)
- Cataracts—less common finding

CAUSES

- Genetic susceptibility
- Infectious (viral) diseases
- Immune-mediated pancreatic β -cell destruction
- Inflammation of the pancreas (known as “pancreatitis”)
- Diseases that increase the likelihood of developing diabetes mellitus (such as increased production of steroids by the adrenal glands [known as “hyperadrenocorticism” or “Cushing’s disease”] and a condition caused by excessive levels of growth hormone, leading to enlargement of bone and soft-tissues in the body [known as “acromegaly”])
- Medications (such as steroids and progestogens [substance capable of producing the effects of the female hormone, progesterone])

RISK FACTORS

- Diestrus (time period following the end of standing heat [when the female is receptive to breeding] and when the female hormone, progesterone, is being secreted by the yellow body of the ovary) in the female dog (known as a “bitch”)



TREATMENT

HEALTH CARE

- Most dogs with uncomplicated diabetes mellitus can be managed as outpatients; they are alert, hydrated, and eating and drinking without vomiting
- Fluid therapy may be needed in some cases

ACTIVITY

- Strenuous activity may lower insulin requirements
- Consistent amount of activity each day is helpful

DIET

- Avoid soft, moist foods because they cause increased glucose (sugar) in the blood following meals (known as “postprandial hyperglycemia”)
- Non-obese dogs—feed a consistent diet that the pet will eat reliably; keep daily caloric intake constant
- Obese dogs—reduce the caloric intake to 60% of the requirement for the animal’s ideal body weight (technique 1) or feed a high-fiber, low-calorie food in an amount similar to what the pet is accustomed to eating (technique 2); try to achieve the desired target weight over 2 to 4 months
- Thin dogs—avoid reduced-calorie diet; starvation worsens ketoacidosis (condition in which levels of acid are increased in the blood due to the presence of ketone bodies secondary to diabetes) and immune function
- Role of fiber—key role is in weight loss and obesity prevention; another benefit may be improved glycemic control; recommended diet is high in fiber, low in fat, and high in complex carbohydrates
- Feed the pet half its daily food every 12 hours to coincide with twice-daily insulin injections or medications administered by mouth to decrease blood glucose or sugar (oral hypoglycemic agents); give animals on once-daily insulin injections half the food with the injection and the remainder in 8 to 10 hours or at the time of peak insulin activity, if that is known
- Discuss the dietary needs and feeding protocol with your pet’s veterinarian

SURGERY

- Intact females (that is, capable of reproducing) should have their ovaries and uterus removed surgically (known as a “spay” or “ovariohysterectomy”), when stable
- Progesterone (a female hormone) secreted during diestrus (time period following the end of standing heat [when the female is receptive to breeding] and when progesterone is being secreted by the yellow body of the ovary) makes management of diabetes mellitus difficult



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Insulin—required for treatment of insulin-dependent diabetes mellitus; frequently utilized as part of management of non-insulin dependent diabetes mellitus; various types, including Vetsulin™ (porcine origin lente insulin); Humulin® N; Novolin® N; PZI VET® (Protamine Zinc Insulin—beef/pork insulin)
- Insulin is administered by injection; your pet’s veterinarian will explain proper handling, storage, and administration of insulin
- Glargine: rarely used in canine diabetics
- Oral administration of medications to decrease blood glucose (hypoglycemic agents) generally is not recommended in canine diabetes



FOLLOW-UP CARE

PATIENT MONITORING

- Glucose curve—“gold standard;” can provide information on insulin effectiveness, duration of action, and nadir (that is, the lowest blood glucose level achieved during dosing interval); used most frequently when establishing initial control, changing insulin type, dose, or frequency, or problem solving the diabetic that is difficult to control
- Glycated proteins—“glycosylated hemoglobin” or “fructosamine;” glucose binds irreversibly to hemoglobin (glycosylated hemoglobin) or albumin (fructosamine); extent of binding or glycosylation directly related to blood glucose concentration over lifespan of protein in blood (5 to 9 weeks for hemoglobin, 1 to 3 weeks for fructosamine); not affected by stress of hospitalization or dietary intake on the day of obtaining blood samples for testing; requires single blood draw, best used for ongoing management of stable diabetic patient;

fructosamine of 400 mg/dl is consistent with adequate glycemic control

- At-home monitoring—urine glucose and/or blood glucose (lancet device for capillary blood from the ear) requires significant owner commitment and compliance; most useful as early indicator of need for reduction in dose with persistent absence of glucose (sugar) in the urine (glucosuria); should never be used as the sole basis for adjustment of insulin; minimal evidence supports the need for the extent of monitoring, frequency of dose adjustments, or exactness of control in pets as is required in the management of human diabetics
- Clinical signs—assess the degree of polyuria/polydipsia, appetite, and body weight; if these are normal, the disease likely is well controlled

PREVENTIONS AND AVOIDANCE

- Prevent or correct obesity
- Avoid unnecessary use of steroids or megestrol acetate in treating various medical conditions

POSSIBLE COMPLICATIONS

- Cataracts can occur, even with good glycemic control
- Seizure or coma with insulin overdose
- Low red-blood cell count (known as “anemia”) and presence of excessive hemoglobin in the plasma (known as “hemoglobinemia”) with severe low levels of phosphorus in the blood (known as “hypophosphatemia”), which can occur after initial insulin therapy; “hemoglobin” is the compound in red-blood cells that carries oxygen to the tissues of the body

EXPECTED COURSE AND PROGNOSIS

- Dogs have permanent disease
- Prognosis with treatment is good; most animals have a normal life span



KEY POINTS

- Discuss daily feeding and medication schedule, home monitoring, signs of low levels of glucose (sugar) in the blood (known as “hypoglycemia”) and what to do if hypoglycemia develops with your pet’s veterinarian
- Understand when it is necessary to call or visit your pet’s veterinarian
- Keep a chart of pertinent information about the pet, such as urine dipstick results, daily insulin dose, and weekly body weights

DIABETES WITH KETOACIDOSIS



BASICS

OVERVIEW

- A true medical emergency; condition secondary to absolute or relative insulin deficiency, characterized by increased levels of glucose (sugar) in the blood (known as “hyperglycemia”), high levels of ketones in the blood (known as “ketonemia”), metabolic acidosis (a condition in which levels of acid are increased in the blood), dehydration, and electrolyte depletion
- “Diabetes” refers to diabetes mellitus (“sugar diabetes”)
- “Diabetes with ketoacidosis” or “diabetic ketoacidosis” is a condition in which levels of acid are increased in the blood due to the presence of ketone bodies secondary to diabetes

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dogs—miniature poodle and dachshund
- Cats—none

Mean Age and Range

- Dogs—mean age, 8.4 years
- Cats—median age, 11 years (range, 1 to 19 years of age)

Predominant Sex

- Dogs—females 1.5 times more likely to develop ketoacidosis than males
- Cats—males 2 times more likely to develop ketoacidosis than females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Increased urination (known as “polyuria”)
- Increased thirst (known as “polydipsia”) or absence or lack of thirst (known as “adipsia”)
- Diminished activity
- Lack of appetite (known as “anorexia”)
- Weakness
- Vomiting
- Sluggishness (lethargy) and depression
- Muscle wasting and weight loss
- Unkempt hair coat
- Rapid breathing (known as “tachypnea”)
- Dehydration
- Thin body condition
- Decreased or low body temperature (known as “hypothermia”)
- Dandruff
- Thickened bowel loops
- Enlarged liver (known as “hepatomegaly”)
- Ketone odor on breath
- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)

CAUSES

- Insulin-dependent diabetes mellitus
- Infection (such as infection of the skin, respiratory tract, urinary tract, prostate gland, kidneys, uterus, or lungs [pneumonia])
- Coexistent disease (such as heart failure, inflammation of the pancreas [known as “pancreatitis”], kidney failure, asthma, cancer)
- Unknown cause (so called “idiopathic disease”)
- Lack of appropriate dosing of medications to treat diabetes mellitus (such as not giving insulin injections on routine schedule)
- Stress
- Surgery

RISK FACTORS

- Any condition that leads to an absolute or relative insulin deficiency
- History of administration of steroids or β -blockers in the treatment of various diseases
- Female dog (known as a “bitch”) in heat or estrus



TREATMENT

HEALTH CARE

- If the animal is bright, alert, and well hydrated, intensive care and intravenous fluid administration are not required; start administration of insulin, offer food, and supply constant access to water; monitor closely for signs of illness (such as lack of appetite [anorexia], lethargy, vomiting)
- Treatment of “sick” diabetic ketoacidotic dog or cat requires inpatient intensive care; this is a life-threatening emergency; goals are to correct the depletion of water and electrolytes, reverse the high levels of ketones and acids in the blood (ketonemia and acidosis), and increase the rate of glucose use by insulin-dependent tissues
- Fluids—necessary to ensure adequate blood volume being pumped by the heart (known as “cardiac output”) and blood flow to the tissues and to maintain blood volume; also helps to reduce blood glucose concentration

DIET

- A low-fat, high-fiber, high-complex-carbohydrate diet is recommended, once the patient is stabilized

MEDICATIONS AND TREATMENT

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Insulin

- Regular insulin is the insulin of choice in the initial treatment of an animal with diabetic ketoacidosis; needed to decrease levels of glucose (sugar) in the blood
- Check blood glucose every 1 to 3 hours to monitor response of blood glucose to insulin
- Monitor urine glucose and ketones daily
- Start administering longer-acting insulin (such as NPH, lente, or ultralente insulin) when the patient is eating, drinking, and no longer receiving intravenous (IV) fluids and levels of ketones are diminished greatly

Potassium Supplementation

- Total body potassium is depleted and treatment (such as fluids and insulin) will further lower serum potassium; potassium supplementation is always necessary
- If possible, check potassium concentration before initiating insulin therapy, to guide supplementation dosage; if serum potassium concentration is extremely low, insulin therapy may need to be delayed (hours) until it increases
- Low levels of potassium in the blood that do not respond to treatment (known as “refractory hypokalemia”) may indicate magnesium depletion, requiring magnesium replacement (using magnesium chloride or magnesium sulfate) as a continuous-rate infusion
- If potassium concentration is unknown, add potassium (40 mEq/L) to the intravenous (IV) fluids; administer intravenous potassium carefully

Dextrose Supplementation

- Must give insulin, regardless of the blood glucose concentration, to correct the ketoacidotic state
- Whenever blood glucose is less than 200 to 250 mg/dl, 50% dextrose should be added to the fluids to produce a 2.5% dextrose solution (increase to 5% dextrose if needed); discontinue dextrose once glucose is maintained above 250 mg/dl
- Do not stop insulin therapy

Bicarbonate Supplementation

- May be considered if patient’s venous blood pH is less than 7.0 or if the total carbon dioxide (CO₂) is less than 11 mEq/L on blood tests (indicates that the animal’s blood is very acidic)

Phosphorus Supplementation

- Pretreatment serum phosphorus usually is normal; however, treatment of ketoacidosis reduces phosphorus, so phosphorus supplementation may be necessary; serum phosphorus concentrations should be checked every 12 to 24 hours once phosphorus supplementation is initiated



FOLLOW-UP CARE

PATIENT MONITORING

- Attitude, hydration, urine output, body weight, and status of heart and lungs should be monitored
- Blood glucose (sugar) levels should be checked every 1 to 3 hours initially; every 6 hours once stable
- Electrolytes (such as potassium, sodium, chloride) should be checked every 4 to 8 hours initially; every 24 hours once stable
- Acid–base status should be checked every 8 to 12 hours initially; every 24 hours once stable

PREVENTIONS AND AVOIDANCE

- Appropriate insulin administration

POSSIBLE COMPLICATIONS

- Low levels of potassium in the blood (hypokalemia)
- Low levels of glucose (sugar) in the blood (hypoglycemia)
- Low levels of phosphorus in the blood (hypophosphatemia)
- Build-up of fluid in the brain (known as “cerebral edema”)
- Build-up of fluid in the lungs (known as “pulmonary edema”)
- Kidney failure
- Heart failure

EXPECTED COURSE AND PROGNOSIS

- Guarded



KEY POINTS

- Diabetic ketoacidosis is a true medical emergency
- Serious medical condition requiring lifelong insulin administration in most patients

CHRONIC DIARRHEA IN CATS



BASICS

OVERVIEW

- A change in the frequency, consistency, and volume of bowel movement (feces) for more than 3 weeks or with a pattern of episodic recurrence
- Can be either small bowel (small intestine) or large bowel (large intestine or colon) diarrhea

SIGNALMENT/DESCRIPTION of ANIMAL

- Cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Underlying disease process determines clinical signs

Small Bowel Diarrhea (involves the small intestines)

- Larger volume of bowel movement (feces) than normal
- Frequency of defecation is mild to moderately above normal (2 to 4 times per day)
- Weight loss
- Increased appetite (known as “polyphagia”) in cases with abnormal digestion or absorption of food (known as “maldigestion” or “malabsorption,” respectively) or increased levels of thyroid hormone (known as “hyperthyroidism”)
- May have black, tarry stools (due to the presence of digested blood; condition known as “melena”); no mucus or red blood in the bowel movement (presence of red blood in the bowel movement known as “hematochezia”)
- No evidence of painful defecation or straining to defecate (known as “tenesmus”) or difficulty in defecating (known as “dyschezia”)
- Vomiting is common

Large Bowel Diarrhea (involves the large intestines or colon)

- Smaller volume of bowel movement (feces) per defecation than normal
- Frequency of defecation significantly higher than normal (greater than 4 times per day)
- No weight loss
- Mucus or red blood in the bowel movement (hematochezia); no evidence of black, tarry stools (melena)
- Painful defecation or straining to defecate (tenesmus) and urgency to defecate
- Difficulty defecating (dyschezia) with rectal or lower colonic disease
- Vomiting in some cats

CAUSES

- Inflammatory bowel disease (“IBD”)—various types, including lymphoplasmacytic enterocolitis, granulomatous enteritis, eosinophilic enteritis/hypereosinophilic syndrome, and idiopathic inflammatory colitis
- Tumor or cancer—lymphoma, adenocarcinoma, mast cell tumor, and polyps
- Blockage or obstruction of the small or large intestines—tumor or cancer, foreign body, inflammatory bowel disease (IBD), folding of one segment of the intestine into another segment (known as “intussusception”), and abnormal narrowing of the intestines (known as a “stricture”)
- Metabolic disorders—increased levels of thyroid hormone (hyperthyroidism), kidney disease, liver disease, diabetes mellitus (“sugar diabetes”)
- Poisons
- Side effect of medications
- Parasites—*Giardia*, *Toxoplasma*, roundworms (*Toxocara cati*, *Toxascaris leonina*), *Cryptosporidium*, *Cystoisospora*, *Tritrichomonas*
- Bacterial infections—*Campylobacter*, *Salmonella*, *Yersinia*, and *Clostridium perfringens*
- Viral infections—feline leukemia virus (FeLV), feline immunodeficiency virus (FIV), and feline infectious peritonitis (FIP)
- Fungal diseases—histoplasmosis, aspergillosis
- Noninflammatory causes of abnormal absorption of food (malabsorption)—dilation of the lymphatic vessels (known as “lymphangiectasia”); condition in which a high number of bacteria are found in the upper small intestine (known as “small intestinal bacterial overgrowth”); diarrhea and other signs caused by absence of a long section of small intestine, usually because of surgical removal (condition known as “short-bowel syndrome”); and ulcers in the upper small intestines (known as “duodenal ulcers”)
- Abnormal digestion of food (maldigestion)—liver disease and syndrome caused by inadequate production and secretion of digestive enzymes by the pancreas (known as “exocrine pancreatic insufficiency”)
- Diet—dietary sensitivity, dietary indiscretion (that is, eating substances that should not be eaten), and diet changes
- Congenital (present at birth) anomalies—short colon; condition in which blood vessels allow blood to flow abnormally between the portal vein (vein that normally carries blood from the digestive organs to the liver) and the body circulation without first going through the liver (known as a “portosystemic shunt”)

RISK FACTORS

- Dietary changes and feeding poorly digestible or high-fat diet



TREATMENT

HEALTH CARE

- Often must be specific for the underlying cause to be successful
- When no definitive diagnosis is possible, treatment with dietary management and metronidazole sometimes results in clinical improvement
- Fluid therapy for dehydration
- Correct electrolyte (such as sodium, potassium, chloride) and acid–base imbalances

DIET

- A bland or hypoallergenic diet may be beneficial

SURGERY

- Biopsy of the stomach, small intestine, and/or large intestine
- Exploratory surgery of the abdomen and surgical biopsy



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medications vary, depending on underlying cause
- A therapeutic trial with fenbendazole or metronidazole often is used to rule out occult *Giardia* infection



FOLLOW-UP CARE

PATIENT MONITORING

- Fecal volume and character, frequency of defecation, and body weight

PREVENTIONS AND AVOIDANCE

- Depend on underlying cause

POSSIBLE COMPLICATIONS

- Dehydration
- Fluid build-up in the abdomen (known as “abdominal effusion”) with intestinal cancer (adenocarcinoma)

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause
- Resolution usually occurs gradually with treatment; if diarrhea does not resolve, consider re-evaluating the diagnosis

CHRONIC DIARRHEA IN DOGS



BASICS

OVERVIEW

- A change in the frequency, consistency, and volume of bowel movement (feces) for more than 3 weeks or with a pattern of episodic recurrence
- Can be either small bowel (small intestine) or large bowel (large intestine or colon) diarrhea

SIGNALMENT/DESCRIPTION of ANIMAL

- Dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Underlying disease process determines clinical signs

Small Bowel Diarrhea (involves the small intestines)

- Larger volume of bowel movement (feces) than normal
- Frequency of defecation is mild to moderately above normal (2 to 4 times per day)
- Weight loss
- Increased appetite (known as “polyphagia”) in cases with abnormal digestion or absorption of food (known as “maldigestion” or “malabsorption,” respectively)
- May have black, tarry stools (due to the presence of digested blood; condition known as “melena”); no mucus or red blood in the bowel movement (presence of red blood in the bowel movement known as “hematochezia”)
- No evidence of painful defecation or straining to defecate (known as “tenesmus”) or difficulty in defecating (known as “dyschezia”)
- May have excessive gas formation in the stomach or intestines (known as “flatulence”) and rumbling or gurgling sounds caused by movement of gas in the intestinal tract (known as “borborygmus”)
- Vomiting in some dogs

Large Bowel Diarrhea (involves the large intestines or colon)

- Smaller volume of bowel movement (feces) per defecation than normal
- Frequency of defecation significantly higher than normal (greater than 4 times per day)
- No weight loss
- Mucus or red blood in the bowel movement (hematochezia); no evidence of black, tarry stools (melena)
- Painful defecation or straining to defecate (tenesmus) and urgency to defecate
- Difficulty defecating (dyschezia) with rectal or lower colonic disease
- Excessive gas formation in the stomach or intestines (flatulence) and rumbling or gurgling sounds caused by movement of gas in the intestinal tract (borborygmus) is variable
- Vomiting is uncommon

CAUSES

Small Bowel Diarrhea (involves the small intestines)

Primary Small Intestinal Disease

- Inflammatory bowel disease (“IBD”)—various types, including lymphoplasmacytic enteritis, granulomatous enteritis, eosinophilic enteritis, immunoproliferative enteropathy of basenjis, and sprue
- Dilation of the lymphatic vessels (known as “lymphangiectasia”)
- Tumor or cancer—such as lymphoma or adenocarcinoma
- Parasites—*Giardia*, roundworms (*Toxocara*), hookworms (*Ancylostoma*), and *Strongyloides*
- Bacterial infection—*Salmonella*, *Clostridium perfringens*
- Fungal disease—histoplasmosis and pythiosis (infection with *Pythium*, a water mold)
- Partial blockage or obstruction—foreign body; folding of one segment of the intestine into another segment (known as “intussusception”); and cancer
- Condition in which a high number of bacteria are found in the upper small intestine (known as “small intestinal bacterial overgrowth”)
- Diarrhea and other signs caused by absence of a long section of small intestine, usually because of surgical removal (condition known as “short-bowel syndrome”)
- Ulcers of the stomach and upper intestines (known as “gastroduodenal ulcers”)

Abnormal Digestion of Food (Maldigestion)

- Syndrome caused by inadequate production and secretion of digestive enzymes by the pancreas (known as “exocrine pancreatic insufficiency”)
- Liver disease

Diet

- Dietary intolerance or allergy
- Gluten-sensitive enteropathy in Irish setters

Metabolic Disorders

- Liver disease; decreased levels of steroids produced by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”); excess levels of urea and other nitrogenous waste products in the blood (known as “uremia”)

Other

- Poisons
- Side effect of medications

Large Bowel Diarrhea (involves the large intestines or colon)

Primary Large Intestinal Disease

- Inflammatory bowel disease (“IBD”)—various types, including lymphoplasmacytic colitis, eosinophilic colitis, histiocytic ulcerative colitis, and granulomatous colitis
- Tumor or cancer—such as benign polyp, lymphoma, adenocarcinoma, leiomyoma, and leiomyosarcoma
- Parasites—whipworms (*Trichuris*), *Giardia*, hookworms (*Ancylostoma*), *Entamoeba*, and *Balantidium*
- Bacterial infections—*Campylobacter*, *Salmonella*, and *Clostridium perfringens*
- Fungal disease—histoplasmosis and pythiosis (infection with *Pythium*, a water mold)
- Infection with algae—*Prototheca*
- Noninflammatory causes (such as folding of one segment of the intestine [the ileum, or lower small intestine] into another segment [the colon]; condition known as an “ileocolic intussusception”)

Diet

- Diet—dietary indiscretion (that is, eating substances that should not be eaten), diet changes, and foreign material (such as bones and hair)
- Fiber

Metabolic Disorders

- Excess levels of urea and other nitrogenous waste products in the blood (uremia)
- Decreased levels of steroids produced by the adrenal glands (hypoadrenocorticism or Addison’s disease)

Other

- Poisons
- Side effect of medications
- Unknown cause (so called “idiopathic disease”)—irritable bowel syndrome

RISK FACTORS

Small Bowel Diarrhea (involves the small intestines)

- Dietary changes and feeding poorly digestible or high-fat diets
- Large-breed dogs, especially German shepherd dogs, have the highest incidence of exocrine pancreatic insufficiency (syndrome caused by inadequate production and secretion of digestive enzymes by the pancreas)
- Pythiosis (infection with *Pythium*, a water mold) occurs most often in young, large-breed dogs living in states bordering the Gulf of Mexico

Large Bowel Diarrhea (involves the large intestines or colon)

- Dietary changes or indiscretion, stress, and psychological factors
- Histiocytic ulcerative colitis (inflammation characterized by a thickened lining of the colon with varying degrees of loss of the superficial lining [known as “ulceration”]; the thickening is due to infiltration of various cells [histiocytes, plasma cells, and lymphocytes] in the layers under the lining) occurs most often in boxers less than 3 years of age
- Pythiosis (infection with *Pythium*, a water mold) occurs most often in young, large-breed dogs living in states bordering the Gulf of Mexico



TREATMENT

HEALTH CARE

- Treat the underlying cause—symptomatic treatment rarely resolves long-term (chronic) diarrhea
- Fecal examinations to identify parasites are often negative in whipworm-infested dogs because of intermittent shedding of eggs; because whipworms are a common cause of diarrhea, deworming with fenbendazole may be performed as a diagnostic aid before pursuing extensive diagnostic tests
- Fluid therapy, if pet is dehydrated
- Consider colloids for pets with low levels of protein in the blood (known as “hypoproteinemia”) that need fluid therapy; “colloids” are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch
- Correct electrolyte (such as sodium, potassium, chloride) and acid–base imbalances
- Some dogs with inflammatory bowel disease or exocrine pancreatic insufficiency (syndrome caused by inadequate production and secretion of digestive enzymes by the pancreas) have secondary small intestinal bacterial overgrowth (condition in which a high number of bacteria are found in the upper small intestine), which must be treated along with the primary disorder

DIET

- Feeding a low-fat, highly digestible diet for 3 to 4 weeks may resolve diarrhea due to dietary intolerance
- Feeding a hypoallergenic diet may be beneficial, if food allergy is suspected

SURGERY

- Biopsy of the stomach, small intestine, and/or large intestine

- Exploratory surgery of the abdomen and surgical biopsy



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medications vary, depending on underlying cause
- In dogs with signs of inflammation of the colon (known as “colitis”), a therapeutic trial with fenbendazole to rule out whipworms may be performed, before pursuing extensive diagnostic testing



FOLLOW-UP CARE

PATIENT MONITORING

- Fecal volume and character, frequency of defecation, and body weight
- In dogs with protein-losing enteropathy (condition in which proteins are lost from the body through the intestines)—serum proteins and clinical signs (fluid build-up in the abdomen [known as “ascites”], under the skin [known as “subcutaneous edema”], in the space between the lungs and chest wall [known as “pleural effusion”])

PREVENTIONS AND AVOIDANCE

- Depend on underlying cause

POSSIBLE COMPLICATIONS

- Dehydration
- Fluid build-up in the abdomen (ascites), under the skin (subcutaneous edema) and/or in the space between the lungs and chest wall (pleural effusion) with low levels of albumin (a type of protein) in the blood (condition known as “hypoalbuminemia”) from protein-losing enteropathy (condition in which proteins are lost from the body through the intestines)

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause
- Complete resolution of signs is not always possible, despite a correct diagnosis and proper treatment
- Resolution of diarrhea usually is gradual after treatment; if it does not resolve with treatment, consider re-evaluating the diagnosis



KEY POINTS

- Complete resolution of signs is not always possible, despite a correct diagnosis and proper treatment

SUDDEN (ACUTE) DIARRHEA



BASICS

OVERVIEW

- Sudden or recent onset of abnormally increased water content and/or solid content in the bowel movements

SIGNALMENT/DESCRIPTION of ANIMAL

- Dogs or cats
- Any animal can suffer from acute diarrhea; kittens and puppies are affected most frequently

SIGNS/OBSERVED CHANGES in the ANIMAL

- Acute diarrhea is usually self-limiting (will resolve quickly), an isolated episode and does not affect the animal in general
- Other cases are mild, do not affect the animal in general, and resolve after a few days
- Sometimes it is sudden (acute) or very sudden (peracute) severe disease; more common in dogs (for example, parvovirus-related diarrhea) than cats
- Patients that are not generally ill have normal body fluid content (hydration) and minimal systemic signs
- Signs of more severe illness (such as vomiting, fever, abdominal pain, blood in the stool [hematochezia], vomiting blood [hematemesis], severe dehydration, weakness, or depression) should prompt more aggressive diagnostic and therapeutic measures
- Fecal accidents, vomiting, changes in fecal consistency and volume, blood or mucus in the feces, or straining to defecate
- Listlessness and lack of appetite (anorexia) may precede diarrhea due to viral enteritis

CAUSES

- Systemic illness may lead to diarrhea as a secondary event
- Dietary indiscretion—eating garbage, nonfood material, or spoiled food
- Dietary changes—sudden changes in amount or type of food, including change in brand of food
- Dietary intolerance—abnormal digestion or absorption of food (maldigestion or malabsorption) of foodstuffs; body “over responds” to a particular ingredient in the food (dietary hypersensitivity)
- Metabolic diseases—such as hypoadrenocorticism (Addison’s disease; disease in which adrenal glands produce inadequate levels of steroids), liver disease, kidney disease, and pancreatic disease can cause acute or chronic diarrhea
- Intestinal blockage (obstruction) or foreign bodies—eating nonfood items (foreign bodies); folding of one segment of the intestine into another segment (known as “intussusception”), or twisting of the intestines and intestinal blood vessels (known as “intestinal or mesenteric volvulus”)
- Unknown cause (known as “idiopathic”)—hemorrhagic gastroenteritis, a specific condition characterized by bloody inflammation of the stomach and intestines and very high packed-cell volume (PCV) caused by the cellular portion of the blood being a high percentage of the blood volume as compared to the fluid portion (a sign of dehydration)
- Infectious causes
 - Viral—parvovirus (canine parvovirus infection and feline panleukopenia), coronavirus, rotavirus, canine distemper virus
 - Bacterial—*Salmonella*, *Campylobacter*, *Clostridium*, *Escherichia coli*
 - Parasitic—hookworms, roundworms, whipworms, strongyles, and tapeworms, *Giardia*., coccidia
 - Rickettsia*—salmon poisoning (*Neorickettsia*)
 - Fungal—Histoplasmosis
- Drugs and toxins—such as heavy metals (example, lead), organophosphates (chemicals found in insecticides), nonsteroidal anti-inflammatory drugs (NSAIDs), steroids, antimicrobials, anthelmintics, cancer drugs, lawn and garden products

RISK FACTORS

- Young dogs and cats present for diarrhea from dietary indiscretion, intussusception, foreign bodies, and infectious causes more often than older patients



TREATMENT

HEALTH CARE

- Depends largely on the severity of illness and underlying cause of the diarrhea
- Patients with mild illness can be treated as outpatients with symptomatic therapy; patients with more severe illness or that fail to respond to symptomatic therapy should be treated more aggressively
- Fluid therapy and correction of electrolyte imbalances is the mainstay of treatment in most cases
- Crystalloid fluid therapy may be administered by mouth (orally), under the skin (subcutaneously), or into a vein (intravenously), as required; can give oral fluids (water or carbohydrate- and electrolyte-containing fluids) to patients that are not vomiting

- Fluid therapy goal is to return the patient to proper body fluid (hydration) status (over 12 to 24 hours) and replace any ongoing losses due to diarrhea and/or vomiting
- Severe body fluid loss can occur with acute diarrhea; aggressive shock fluid therapy may be necessary
- Potassium supplementation may be necessary; patients with severely low levels of potassium in their blood (known as “hypokalemia”) may require more aggressive potassium supplementation

ACTIVITY

- Based on the severity of illness, underlying cause of the diarrhea, and necessary treatment (medical, surgical or both)

DIET

- Patients with mild illness that are not vomiting—a period of fasting (12 to 24 hours) often is followed by a bland diet, such as boiled rice and chicken or a commercial therapeutic diet (for example, Hill’s Prescription Diet® i/d®, Purina Veterinary Diets® EN®)
- Diet recommendations are based on the severity of illness and underlying cause of the diarrhea
- Limit exposure to garbage, foods other than the patient’s normal diet, and potential foreign bodies

SURGERY

- Treatment of intestinal intussusception (the folding of one segment of the intestine into another segment)
- Treatment of intestinal blockage (obstruction), intestinal twisting (intestinal or mesenteric volvulus), or removal of foreign bodies



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antidiarrheal drugs can be classified as drugs that change the movements of the intestinal tract (known as “motility-modifying drugs”), drugs that decrease secretions in the intestinal tract (known as “antisecretory drugs”), or drugs that coat or protect the lining of the intestines (known as “intestinal protectants”)
- Motility-modifying drugs generally operate by increasing the movement of certain segments of the intestinal tract (known as “segmental motility”) and thus increasing the time for materials within the intestinal tract to move through it (that is, it increases transit time; an example of this type of motility-modifying drug is loperamide) or by decreasing forward movement or motility of the intestinal tract (examples of this type of motility-modifying drug are the anticholinergics); these medications are not necessary in mild disease, as it is generally self-limiting
- In severe disease, the veterinarian will administer fluids
- Acute diarrhea that does not resolve with antidiarrheal drugs merits further investigation
- Anticholinergics (such as atropine, propantheline) can produce a generalized lack of movement in the intestine, leading to a condition known as “ileus,” because the drugs decrease segmental and peristaltic motion; this decrease in tone can increase the severity of diarrhea in some patients
- Antisecretory drugs (such as opiates, anticholinergics, chlorpromazine, and salicylates) are used to decrease the volume of fluid in the feces
- Bismuth subsalicylate may be of some benefit because of the antisecretory properties of salicylate; cats can be sensitive to the drug
- Intestinal protectants generally are not helpful in patients with acute diarrhea and have not been shown to change intestinal fluid or electrolyte loss
- Treatment for intestinal parasites if parasites are suspected (empirical treatment) or if parasites or their eggs are seen on analysis of feces (definitive treatment)
- Antibiotic therapy is probably unnecessary for most cases of mild illness and may actually cause diarrhea; patients with bacteria-caused inflammation of the intestines (enteritis), severe illness, low white blood-cell counts (known as “leukopenia”), or suspected breakdown of the gastrointestinal mucosal barrier (as evidenced by blood in the feces) should be treated with broad-spectrum antibiotics



FOLLOW-UP CARE

PATIENT MONITORING

- Most acute diarrhea resolves within a few days
- If clinical signs persist, additional diagnostics and treatments may be necessary
- Recheck stool samples in those patients that had parasites identified by fecal analysis
- Monitor for intussusception in patients with acute diarrhea, especially young dogs with parvoviral diarrhea and parasitism

PREVENTIONS AND AVOIDANCE

- Puppies and kittens should be vaccinated against infectious causes of diarrhea (such as parvovirus) and treated for intestinal parasites per recommendations from your pet’s veterinarian
- Routine stool sample analysis for intestinal parasites
- Limit exposure to garbage, foods other than the patient’s normal diet, and potential foreign bodies

POSSIBLE COMPLICATIONS

- Intussusception, thought to be associated with increased intestinal motility
- *Campylobacter* enteritis is contagious to people
- Some strains of *Giardia* may be contagious to people
- Roundworm larvae can migrate through the body causing visceral larval migrans and hookworm larvae can migrate under the skin causing cutaneous larval migrans in people, particularly children

EXPECTED COURSE AND PROGNOSIS

- Most acute diarrhea resolves within a few days
- Determined by the severity of illness, underlying cause of the diarrhea, and necessary treatment (medical, surgical or both)



KEY POINTS

- Sudden or recent onset of abnormally increased water content and/or solid content in the bowel movements
- Any animal can suffer from acute diarrhea; kittens and puppies are affected most frequently
- Acute diarrhea is usually self-limiting (will resolve quickly)
- Signs of more severe illness (such as vomiting, fever, abdominal pain, blood in the stool [hematochezia], vomiting blood [hematemesis], severe dehydration, weakness, or depression) should prompt more aggressive diagnostic and therapeutic measures

DISKOSPONDYLITIS

(INFLAMMATION OF THE INTERVERTEBRAL DISKS AND ADJACENT BONE OF THE SPINE)



BASICS

OVERVIEW

- A bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies)
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine

GENETICS

- No definite predisposition identified
- An inherited immunodeficiency (inability to develop a normal immune response) has been detected in a few cases

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs; rare in cats

Breed Predisposition

- Large- and giant-breed dogs, especially German shepherd dogs and Great Danes

Mean Age and Range

- Mean age—4 to 5 years of age
- Range—5 months to 12 years of age

Predominant Sex

- Males outnumber females by approximately 2:1

SIGNS/OBSERVED CHANGES in the ANIMAL

- Onset usually relatively sudden (acute); some patients have mild signs for several months before examination
- Pain—difficulty rising, reluctance to jump, and stilted gait are most common signs
- Wobbly gait (known as “ataxia”)
- Weight loss and lack of appetite (anorexia)
- Lameness
- Draining lesions or tracts
- Single or multiple areas of pain along the spine in more than 80% of patients
- Any disk space of the spine may be affected; the lower back (lumbosacral space) is involved most commonly
- Weakness (known as “paresis”) or paralysis, especially in chronic, untreated cases
- Fever in approximately 30% of patients

CAUSES

- Bacterial infection—*Staphylococcus intermedius* is the most common bacterial cause; others include *Streptococcus*, *Brucella canis*, and *E. coli*, but virtually any bacteria can be causative
- Fungal infection—*Aspergillus*, *Paecilomyces*, and *Coccidioides immitis*
- Grass-awn migration often is associated with mixed infections, especially *Actinomyces*; tends to affect the second through fourth lumbar (L2–L4) disk spaces and vertebrae
- Other causes—surgery, bite wounds

RISK FACTORS

- Urinary tract infection
- Infection in gums around the teeth (periodontal disease)
- Bacterial heart infection (endocarditis)
- Skin infection (pyoderma)
- Immunodeficiency (condition in which the animal has an inability to develop a normal immune response)



TREATMENT

HEALTH CARE

- Outpatient—mild pain managed with medication
- Inpatient—severe pain or progressive nervous system deficits and signs require intensive care and monitoring
- Patients that cannot walk (nonambulatory patients)—keep on a clean, dry, well-padded surface to prevent the development of “bed sores” (known as “decubital ulceration”)

ACTIVITY

- Restricted

DIET

- Normal

SURGERY

- Surgical scrapping (known as “curettage”) of a single affected disk space—occasionally necessary for patients that are not responsive to antibiotic therapy
- Goals—remove infected tissue; obtain tissue for bacterial culture and sensitivity and for microscopic (histologic) evaluation
- Relieve pressure on the spinal cord (known as “decompression”) by surgically removing a portion of bone in one or more vertebrae (surgical procedures are known as “hemilaminectomy” or “dorsal laminectomy”)—indicated when the animal has substantial nervous system problems and spinal cord compression is evident on myelography (a special type of X-rays, where contrast dye is injected into the space around the spinal cord to allow visualization of the spinal cord); when no improvement is seen with antibiotic therapy; also perform surgical scrapping (curettage) of the infected disk space; it may be necessary to perform some type of surgical stabilization of the spine



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antibiotics

- Selection based on results of blood cultures and sensitivity testing and *Brucella canis* serum testing (serology)
- Negative culture and serology results—assume causative organism is *Staphylococcus*; treat with a cephalosporin (for example, cefadroxil) for 8 to 12 weeks
- Suddenly progressive signs or substantial nervous system deficits—initially treat with injectable antibiotics (for example, cefazolin)
- Brucellosis—treated with tetracycline and streptomycin or enrofloxacin
- Initial therapy—cephradine; cloxacillin
- Patients that do not respond to initial antibiotic therapy (known as “refractory patients”)—clindamycin, enrofloxacin, orbifloxacin

Analgesics

- Signs of severe pain—treated with an analgesic (for example, oxymorphone)
- Taper dosage after 3 to 5 days to gauge effectiveness of antibiotic therapy



FOLLOW-UP CARE

PATIENT MONITORING

- Reevaluate after 5 days of therapy
- No improvement in pain, fever, or appetite—reassess therapy; consider a different antibiotic, aspirate the affected disk space by passing a sterile needle through the skin and using a syringe (known as “percutaneous aspiration”) or by surgery
- Improvement—evaluate clinically and with X-rays every 4 weeks.

PREVENTIONS AND AVOIDANCE

- Early identification of predisposing causes and prompt diagnosis and treatment—help reduce progression of clinical signs (such as fever, pain) and nervous system signs

POSSIBLE COMPLICATIONS

- Spinal cord compression secondary to development of excessive bone and scar tissue
- Fracture or dislocation of the backbone (vertebra)
- Inflammation of the membranes of the spinal cord (known as “meningitis”) or inflammation of the spinal cord and its surrounding membranes (known as “meningomyelitis”)
- Abscess involving the dura mater (epidural abscess)

EXPECTED COURSE AND PROGNOSIS

- Recurrence is common if antibiotic therapy is stopped prematurely (before 8 to 12 weeks of treatment)
- Some patients require prolonged therapy (1 year or more)
- Prognosis—depends on causative organism and degree of spinal cord damage
- Mild or no nervous system dysfunction (dogs)—usually respond within 5 days of starting antibiotic therapy
- Substantial weakness (paresis) or paralysis (dogs)—prognosis guarded; may note gradual resolution of nervous system dysfunction after several weeks of therapy; treatment warranted
- *Brucella canis*—signs usually resolve with therapy; infection may not be eradicated; recurrence common



KEY POINTS

- A bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies)
- Recurrence is common if antibiotic therapy is stopped prematurely (before 8 to 12 weeks of treatment)
- Response to treatment is very important in determining need for further diagnostic or therapeutic procedures
- Immediately contact your pet's veterinarian if clinical signs progress or recur or if nervous system deficits develop

DISSEMINATED INTRAVASCULAR COAGULOPATHY

(A BLOOD-CLOTTING DISORDER)



BASICS

OVERVIEW

- A bleeding problem in which clotting factors are activated and clotting factors and platelets are used up, leading to bleeding; “clotting factors” are components in the blood involved in the clotting process—the clotting factors are identified by Roman numerals, I through XIII; “platelets” are normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- A complex blood-clotting defect with enhanced clotting (known as “coagulation”) that uses up the body’s clotting factors and causes the formed clots to dissolve (known as “fibrinolysis”) secondary to severe generalized (systemic) disease
- Commonly known as “DIC”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats, more common in dogs

Mean Age and Range

- Depend on the primary, underlying disease

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary with the primary disease and with disseminated intravascular coagulopathy (DIC)-associated organ dysfunction
- Multiple small bruises (known as “petechiae”) and bleeding from sites where blood was drawn or where intravenous (IV) catheters have been placed, bleeding from the moist tissues of the body (known as “mucosa”), or bleeding in body cavities

CAUSES

- Condition in which the stomach dilates with gas and/or fluid (known as “gastric dilatation”), and subsequently rotates around its short axis (known as “volvulus”)—condition known as “gastric dilatation-volvulus” or “bloat”
- Heart failure
- Heartworm disease
- Heat stroke
- Breakdown of red-blood cells (known as “hemolysis”), especially caused by the immune system breaking down the red-blood cells (known as “immune-mediated hemolysis”)
- Inflammation of the stomach and intestines, characterized by the presence of blood (known as “hemorrhagic gastroenteritis”)
- Generalized (systemic) infectious diseases (especially those that cause bacterial toxins to accumulate in the blood [known as “endotoxemia”])
- Liver disease, if severe (especially the disease in which fats and lipids [compounds that contain fats or oils] accumulate in the liver in cats [condition known as “feline hepatic lipidosis”])
- Cancer, especially hemangiosarcoma, breast cancer (mammary carcinoma), and lung cancer (pulmonary adenocarcinoma) in dogs and lymphoma in cats
- Nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin)
- Inflammation of the pancreas (known as “pancreatitis”)
- Shock, low levels of oxygen in the blood and tissues (known as “hypoxia”), conditions in which relative levels of acid are increased in the blood (known as “acidosis”)
- Low platelet or thrombocyte counts caused by the immune system destroying the platelets (known as “immune-mediated thrombocytopenia”); “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding; if they accumulate in a blood vessel, they may lead to a blood clot (known as “thrombosis”)
- Trauma
- Venom

RISK FACTORS

- Vary with cause



TREATMENT

HEALTH CARE

- Requires intensive inpatient treatment
- Aggressive treatment of the primary disease is essential
- Maintain tissue blood flow and oxygen levels using fluids, transfusions and oxygen therapy
- Restore depleted clotting factors by blood/plasma transfusions
- Prevent further clotting within a blood vessel (thrombosis)

ACTIVITY

- Is limited by the disease severity

SURGERY

- Related to primary, underlying disease



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Successful medical treatments are based mostly on experience and traditionally use heparin to effect (as determined by clinical improvement of the patient and blood test results)
- Heparin dosage depends on severity of signs and laboratory test results
- Plasma or blood transfusions often are needed for heparin to be effective
- Low molecular weight heparin: many forms with variable activity are available; fewer complications reported, but very expensive



FOLLOW-UP CARE

PATIENT MONITORING

- Clinical improvement and stopping bleeding are key positive findings
- Daily blood testing is warranted in severe cases to identify positive or negative trends; less frequent blood testing may suffice in milder cases
- Some blood tests (coagulation times and fibrinogen) often return to normal more rapidly than other blood tests (fibrinogen degradation products [FDPs] and platelet counts)

PREVENTIONS AND AVOIDANCE

- Related to primary, underlying disease

POSSIBLE COMPLICATIONS

- Vary with underlying cause
- Death

EXPECTED COURSE AND PROGNOSIS

- Animals with the clotting disorder, disseminated intravascular coagulopathy (DIC), have a high rate of death because of the serious nature of the primary, underlying diseases



KEY POINTS

- The associated generalized (systemic) disease is life-threatening
- Disseminated intravascular coagulopathy (DIC) is a serious complication of a severe disease process in the animal

CANINE DISTEMPER



BASICS

OVERVIEW

- Contagious disease that appears suddenly (acute) or over a moderate amount of time (known as “subacute”), characterized by fever and a variety of signs involving the eyes, central nervous system, and respiratory, urogenital, and gastrointestinal tracts; often a fatal disease
- Caused by the canine distemper virus
- Affects many different species of the order Carnivora; mortality rate varies greatly among species

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Most species of the order Carnivora—including dogs, fox, wolves, hyenas, weasels, ferrets, mink, raccoons, skunks, and civets
- Large cats in Californian zoos and in Tanzania

Mean Age and Range

- Young animals are more susceptible to infection than are adults

SIGNS/OBSERVED CHANGES in the ANIMAL

- Fever—first fever occurs 3 to 6 days after infection, may go undetected; second fever several days later (and intermittent thereafter), usually associated with discharge from the nose and eyes, depression, and lack of appetite (known as “anorexia”)
- Gastrointestinal and/or respiratory signs follow, often enhanced by secondary bacterial infection
- Central nervous system signs—occur in many infected dogs; often, but not always, after generalized (systemic) disease; depends on the virus strain; either sudden (acute) gray or white matter disease (“gray matter” is the nerve tissue of the brain and spinal cord that contains the nerve cell bodies; “white matter” is the part of the brain and spinal cord that contains nerve fibers covered with myelin, a fatty covering that increases conduction of nerve impulses)
- Gray-matter disease—affects the brain and spinal cord; may cause inflammation of the meninges (the membranes covering the brain and spinal cord; inflammation of the meninges known as “meningitis”), seizures, stupor, hysteria, and wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”); dogs may die in 2 to 3 weeks, recover (associated with prompt immune response), or progress to white-matter disease
- White-matter disease—variable signs of disease involving multiple locations of the central nervous system; commonly see weakness and wobbly, incoordinated or “drunken” appearing gait or movement (ataxia) secondary to spinal cord disease; occasionally may see twitching or contraction of a group of muscles (known as “myoclonus”); some dogs die 4 to 5 weeks after initial infection; some dogs may recover with minimal central nervous system injury
- Inflammation of the optic nerve (the nerve that runs from the back of the eye to the brain; condition known as “optic neuritis”) and lesions in the back of the eye (known as the “retina”) may occur
- Hardening of the footpads (known as “hyperkeratosis”) and nose—some virus strains; but relatively uncommon
- Abnormal development of the enamel layer of the teeth (known as “enamel hypoplasia”) after neonatal infection is common

CAUSES

- Canine distemper virus (closely related to the measles virus and the seal and dolphin distemper viruses)
- Incompletely altered, modified live canine distemper virus vaccines (rare)

RISK FACTORS

- Contact of animals that have not been vaccinated or have not responded to vaccinations with animals that are infected with canine distemper virus (dogs or wild carnivores)



TREATMENT

HEALTH CARE

- Inpatient treatment in isolation, to prevent infection of other dogs
- Supportive treatment
- Intravenous fluids—cases with lack of appetite (anorexia) and diarrhea
- Once fevers and secondary bacterial infections are controlled, patients usually begin to eat again
- Carefully clean away discharges from the nose and eyes

ACTIVITY

- Limited

DIET

- Depends on the extent of gastrointestinal involvement



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antiviral drugs—none known to be effective in treating canine distemper viral infections
- Antibiotics—to reduce secondary bacterial infection, because canine distemper virus decreases the ability of the animal to develop a normal immune response (known as “immunosuppression”)
- Medication to control seizures (known as “anticonvulsant therapy”)—phenobarbital, potassium bromide



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor for signs of pneumonia or dehydration from diarrhea in the sudden (acute) phase of the disease
- Monitor for central nervous system signs, because seizures generally follow

PREVENTIONS AND AVOIDANCE

- Routine vaccination against canine distemper virus is key to prevention and avoidance; series of vaccinations administered initially followed by periodic booster vaccinations, as directed by your pet’s veterinarian
- Avoid infection of puppies by isolation to prevent infection from wildlife (such as raccoons, fox, skunks) or from canine distemper virus-infected dogs

POSSIBLE COMPLICATIONS

- Secondary bacterial infections, frequently involve the respiratory and gastrointestinal systems
- Possibility of occurrence of central nervous system signs for 2 to 3 months after discharge from the eyes and nose has subsided
- Seizures
- Death

EXPECTED COURSE AND PROGNOSIS

- Depend on the strain of virus and the individual host response—animal may be infected, but have no signs of disease (known as a “subclinical infection”) or have signs of disease involving various areas of the body; the infection may be fatal or non-fatal
- Mild central nervous system signs—patient may recover; twitching or contraction of a group of muscles (myoclonus) may continue for several months or indefinitely
- Death—2 weeks to 3 months after infection; mortality rate approximately 50%
- Euthanasia—owner may elect euthanasia, if or when nervous system signs develop; indicated when repeated seizures occur
- Fully recovered dogs are not carriers, as they do not shed canine distemper virus



KEY POINTS

- Mortality rate is about 50%
- Dogs that appear to recover from early signs (such as discharge from the eyes and nose) may later develop fatal central nervous system signs
- Fully recovered dogs are not carriers, as they do not shed canine distemper virus
- Routine vaccination against canine distemper virus is key to prevention and avoidance; series of vaccinations administered initially followed by periodic booster vaccinations, as directed by your pet’s veterinarian

DIFFICULTY SWALLOWING (DYSPHAGIA)



BASICS

OVERVIEW

- Difficulty swallowing, resulting from the inability to grasp (prehend), form, and move a mass of chewed food (bolus) through the back of the mouth and throat (known as the “oropharynx”) into the esophagus (the tube running from the oropharynx to the stomach)

SIGNALMENT/DESCRIPTION of ANIMAL

- Dogs and cats
- Congenital (present at birth) disorders that cause difficulty swallowing (such as cricopharyngeal achalasia [a condition of the nerves and muscles in the upper esophagus, in which the muscles do not relax adequately to allow swallowing] and cleft palate) usually are diagnosed in animals less than 1 year of age
- Acquired (develop after birth) disorders that cause difficulty swallowing related to the throat or pharynx (known as “pharyngeal dysphagia”) are more common in older patients

SIGNS/OBSERVED CHANGES in the ANIMAL

- Drooling, gagging, weight loss, ravenous appetite, repeated attempts at swallowing, swallowing with the head in an abnormal position, coughing (due to aspiration), regurgitation, painful swallowing, and occasionally lack of appetite (anorexia) are possible
- Foreign bodies cause sudden (acute) signs of difficulty swallowing; pharyngeal problems may cause long-term (chronic) and intermittent signs of difficulty swallowing
- Observe the patient eating; this may localize the abnormal phase of swallowing
- Modified eating behavior (such as eating with head tilted to one side and throwing head back while eating)
- Paralysis of the lower jaw (mandible) or tongue, dental disease, abnormalities of the muscles on the top of the head (masticatory muscles), inability to open the mouth, and food packed in the cheek area of the mouth, without the presence of saliva suggest problems with the mouth (known as “oral dysphagia”)
- Normal grasping (prehension) of the food with repeated attempts at swallowing, while repeatedly bending (flexing) and straightening (extending) the head and neck; excessive chewing, and gagging suggest problems with the throat (known as “pharyngeal dysphagia”); saliva-coated food retained in the cheek area of the mouth, a diminished gag reflex, and nasal discharge from aspiration also may be seen
- Repeated, nonproductive efforts to swallow, gag, and cough, then forcibly bring food back up (regurgitate) immediately after swallowing with normal gag reflex and grasping (prehension) of food; emaciation suggest problems in the upper esophagus or cricopharyngeal area (known as “cricopharyngeal dysphagia”)

CAUSES

- Anatomic or mechanical lesions include inflammation of the throat or pharynx (for example, abscess, inflammatory polyps, and oral eosinophilic granuloma [a mass or nodular lesion containing a type of white blood cell, called eosinophils], enlarged lymph nodes behind the throat (known as “retropharyngeal lymphadenomegaly”), cancer, foreign body in the throat or behind the throat, cystic mass under the tongue or base of the mouth (known as a “sialocele”), disorders of the joint of the lower jaw (known as “temporomandibular joint [TMJ] disorders”), mandibular fracture, cleft palate, and trauma to the throat
- Pain as a result of dental disease (for example, tooth fractures and abscess), mandibular trauma, inflammation of the mouth (stomatitis), inflammation of the tongue (glossitis), and inflammation of the throat (pharyngitis) may disrupt normal swallowing
- Disorders of the nerves and muscles involved in swallowing include cranial nerve deficits and inflammation of the muscles of chewing (known as “masticatory muscle myositis”)
- Weakness (paresis) or paralysis of the muscles of the throat or pharynx can be caused by infectious agents (such as *Toxoplasma* and *Neospora*), immune-mediated inflammation of several muscles (known as “polymyositis”), muscular dystrophy, various nerve diseases, and disorders involving the area between the muscle and nerves (known as “myoneural junction disorders,” such as myasthenia gravis, tick paralysis, and botulism)
- Rabies can cause difficulty swallowing by affecting the brain stem and peripheral nerves
- Other central nervous system disorders, especially those involving the brain stem

RISK FACTORS

- Many of the causative neuromuscular conditions have breed predispositions



TREATMENT

HEALTH CARE

- Primary treatment is directed at the underlying cause for the difficulty in swallowing
- Patients with oral dysphagia may be able to swallow if a small sphere of food is placed carefully in the back of the mouth; other

patients may find a gruel that can be lapped easier to swallow; take care to avoid aspiration when feeding orally

- Elevating the head and neck may make swallowing easier for patients with problems in the throat or upper esophagus leading to difficulty swallowing (pharyngeal or cricopharyngeal dysphagia) and help prevent aspiration of food

DIET

- Nutritional support is important for all patients having difficulty swallowing
- Multiple, small meals or gruel may allow easier swallowing

SURGERY

- If nutritional requirements cannot be met orally, a feeding tube may be implanted surgically into the stomach
- Surgical removal of a mass or foreign body may be curative or temporarily improve difficulty in swallowing
- Surgically cutting the muscle in the upper esophagus (known as “cricopharyngeal myotomy”) may benefit patients with cricopharyngeal dysphagia



MEDICATIONS

- Dysphagia or difficulty swallowing generally is not immediately life threatening; medications should be directed at the underlying cause



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor daily for signs of aspiration pneumonia (such as depression, fever, whitish to yellowish discharge from the nose, coughing, and difficulty breathing [dyspnea])
- Check body condition and hydration status daily; if oral nutrition does not maintain the animal’s weight and fluid needs, use tube feeding

PREVENTIONS AND AVOIDANCE

- Feeding multiple, small meals with the patient in an upright position and maintaining this position for 10 to 15 minutes after feeding helps prevent aspiration of food

POSSIBLE COMPLICATIONS

- Aspiration pneumonia is a common complication with swallowing disorders
- Consider rabies in any patient with difficulty swallowing (dysphagia), especially if the animal’s rabies vaccination status is unknown or questionable or if the animal has been exposed to a potentially rabid animal

EXPECTED COURSE AND PROGNOSIS

- Depends on the underlying cause



KEY POINTS

- Primary treatment is directed at the underlying cause of the difficulty in swallowing
- Nutritional support is important for all patients having difficulty swallowing
- Feeding multiple, small meals with the patient in an upright position and maintaining this position for 10 to 15 minutes after feeding helps prevent aspiration of food
- Aspiration pneumonia is a common complication with swallowing disorders

ELBOW DYSPLASIA

(ABNORMAL DEVELOPMENT OF THE ELBOW)



BASICS

OVERVIEW

- “Dysplasia” is the medical term for abnormal development
- “Elbow dysplasia” is a series of four developmental abnormalities that lead to malformation and degeneration of the elbow joint
- Most common cause for elbow pain and lameness
- One of the most common causes for forelimb lameness in large-breed dogs

GENETICS

- Inherited disease
- High heritability

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Large- and giant-breed dogs—Labrador retrievers, rottweilers, Golden retrievers, German shepherd dogs, Bernese mountain dogs, chow chows, bearded collies, Newfoundlands

Mean Age and Range

- Age at onset of clinical signs—typically 4 to 10 months
- Age at diagnosis—generally 4 to 18 months
- Onset of signs related to progressive and permanent deterioration of joint cartilage (known as “degenerative joint disease” or “DJD”)—any age

Predominant Sex

- Bone fragment located at the inner surface of the upper ulna (one of the bones of the foreleg), just below the elbow joint (known as “fragmented medial coronoid process”)—males more likely to be affected than females
- Failure of the bones to fuse in the elbow of the growing dog (known as “united anconeal process”) and abnormal development of bone and cartilage, leading to a flap of cartilage within the joint (known as “osteochondritis dissecans” or “OCD”)—none established

SIGNS/OBSERVED CHANGES in the ANIMAL

- Not all affected dogs have signs when young
- Sudden (acute) episode of elbow lameness due to advanced degenerative joint disease (progressive and permanent deterioration of joint cartilage) changes in a mature patient—common
- Intermittent or persistent forelimb lameness—worsened by exercise; progresses from stiffness seen only after rest
- Pain when extending or flexing the elbow
- Affected limb—tendency to be held away from the body (known as “abduction”)
- Fluid build-up in the joint (known as “joint effusion”)
- Grating detected with joint movement (known as “crepitus”)—may be detected with advanced degenerative joint disease (progressive and permanent deterioration of joint cartilage)
- Diminished range of motion

CAUSES

- Genetic
- Developmental
- Nutritional

RISK FACTORS

- Rapid growth and weight gain
- High-calorie diet



TREATMENT

HEALTH CARE

- Surgery—controversial, but recommended for most patients
- Cold packing the elbow joint immediately following surgery to help decrease swelling and control pain; perform at least 5 to 10 minutes every 8 hours for 3 to 5 days, or as directed by your pet's veterinarian
- Range-of-motion exercises—beneficial until the patient can bear weight on the limb(s)

ACTIVITY

- Restricted for all patients postoperatively
- Following surgery—limit activity for a minimum of 4 weeks; encourage early, active movement of the affected joint(s)

DIET

- Weight control—important for decreasing the load and stress on the affected joint(s)
- Restricted weight gain and growth in young dogs—may decrease incidence and severity

SURGERY

- Severity of degenerative joint disease (progressive and permanent deterioration of joint cartilage) and advanced age of patient—negatively influence outcome
- A variety of surgical techniques are possible, depending on type of elbow abnormality
- Using a special lighted instrument called an “arthroscope” (general term for procedure is “arthroscopy”) to allow the surgeon to see inside the joint may be used to diagnose and to treat elbow abnormalities



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No medication promotes healing of bone and/or cartilage fragments
- Nonsteroidal anti-inflammatory drugs (NSAIDs)—minimize pain, decrease inflammation, symptomatically treat associated degenerative joint disease (progressive and permanent deterioration of joint cartilage); examples include carprofen, deracoxib, etodolac, meloxicam, tepoxalin
- Medications intended to slow the progression of arthritic changes and protect joint cartilage (known as “chondroprotective drugs”), such as polysulfated glycosaminoglycans, glucosamine, and chondroitin sulfate—may help limit cartilage damage and degeneration; may help alleviate pain and inflammation



FOLLOW-UP CARE

PATIENT MONITORING

- Yearly examinations—recommended to assess progression and deterioration of joint cartilage (degenerative joint disease or DJD)

PREVENTIONS AND AVOIDANCE

- Discourage breeding of affected animals.
- Do not repeat dam–sire breedings that result in affected offspring

POSSIBLE COMPLICATIONS

- Degenerative joint disease (progressive and permanent deterioration of joint cartilage)

EXPECTED COURSE AND PROGNOSIS

- Progression of degenerative joint disease—expected
- Prognosis—fair to good for all forms



KEY POINTS

- Elbow dysplasia is a genetic disease
- Potential exists for progressive and permanent deterioration of joint cartilage (degenerative joint disease or DJD)
- Excessive intake of nutrients that promote rapid growth has an influence on the development of elbow dysplasia; therefore, restricted weight gain and growth in young dogs may decrease the incidence of elbow dysplasia

HIP DYSPLASIA



BASICS

OVERVIEW

- The failure of normal development (known as “malformation”) and gradual deterioration, leading to loss of function, (known as “degeneration”) of the hip joints (known as the “coxofemoral joints”)
- The hip joint is composed of the “ball” (known as the “femoral head”) and the “socket” (known as the “acetabulum”)

GENETICS

- Complicated pattern of inheritance, multiple genes involved (known as “polygenetic transmission”)
- Development of hip dysplasia determined by an interaction of genetic and environmental factors
- Some breeds are more likely to have the genes for hip dysplasia than other breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs—one of the most common skeletal diseases seen in dogs
- Cats—incidence is significantly lower than in dogs

Breed Predilection

- Large-breed dogs—St. Bernards; German shepherd dogs; Labrador retrievers; golden retrievers; rottweilers
- Smaller breed dogs—may be affected; less likely to show clinical signs
- Cats—more commonly affects purebred cats; reportedly affects approximately 18% of Maine coon cats

Mean Age and Range

- Begins in the immature dog
- Clinical signs—may develop after 4 months of age or may develop later due to osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage)

Predominant Sex

- Dogs—none
- Cats—more common in female cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on the degree of joint looseness or laxity; degree of osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage); and duration of the disease
- Early disease—signs related to joint looseness or laxity
- Later disease—signs related to joint degeneration and osteoarthritis
- Decreased activity
- Difficulty rising
- Reluctance to run, jump, or climb stairs
- Intermittent or persistent hind-limb lameness—often worse after exercise
- “Bunny-hopping” or swaying gait
- Narrow stance in the hind limbs
- Painful hip joints
- Joint looseness or laxity—characteristic of early disease; may not be seen in long-term (chronic) hip dysplasia due to arthritic changes in the hip joint
- Grating detected with joint movement (known as “crepitus”)
- Decreased range of motion in the hip joints
- Loss of muscle mass (known as “atrophy”) in thigh muscles
- Enlargement (known as “hypertrophy”) of shoulder muscles; occurs because dog puts more weight on front legs as it tries to avoid weight on its hips, leading to extra work for the shoulder muscles and subsequent enlargement

CAUSES

- Genetic susceptibility for hip looseness or laxity
- Rapid weight gain, nutrition level, and pelvic-muscle mass—influence development and progression of hip dysplasia

RISK FACTORS

- Overweight and poor muscle tone



TREATMENT

HEALTH CARE

- May treat with conservative medical therapy or surgery
- Outpatient, unless surgery is performed
- Depends on the patient's size, age, and intended function; severity of joint looseness or laxity; degree of osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage); veterinarian's preference for treatment; and financial considerations of the owner
- Physiotherapy (passive joint motion)—decreases joint stiffness; helps maintain muscle integrity
- Swimming (hydrotherapy)—excellent form of physical therapy; encourages joint and muscle activity, without increasing the severity of joint injury

ACTIVITY

- As tolerated by the animal
- Swimming—recommended to maintain joint mobility, while minimizing weight-bearing activities

DIET

- Weight control—important; decreases the pressure applied to the painful joint as the animal moves; minimize weight gain associated with reduced exercise
- Special diets designed for rapidly growing large-breed dogs—may decrease severity of hip dysplasia

SURGERY

Triple Pelvic Osteotomy ("TPO")

- Corrective orthopedic surgical procedure; designed to re-establish corresponding surfaces (known as "congruity") between the "ball" (femoral head) and the "socket" (acetabulum) making up the hip joint
- Immature patient (6 to 12 months of age)
- Rotate the "socket" (acetabulum)—to improve coverage of the "ball" (femoral head); correct forces acting on the joint; minimize the progression of osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage); may allow development of a more normal joint, if performed early (before joint deterioration or degeneration develops)

Juvenile Pubic Symphysiodesis (surgical procedure to fuse the pubis [part of the pelvis] bones together)

- The pelvis develops from matching bones on the right- and left-side of the body; the area where the two sides meet is composed of cartilage and is called a "symphysis;" the pubis is a part of the pelvis; the surgical procedure fuses the pubic symphysis at an early age (using electrocautery)
- Causes the "socket" (acetabulum) to better cover the "ball" (femoral head)
- Improves relationship of corresponding surfaces of the joint and joint stability—similar effects as TPO, without surgical metal implants
- Minimal postoperative problems; easy to perform—must be performed very early (3 to 4 months of age) to achieve effect; minimal effect achieved if performed after 6 months of age

Total Hip Replacement

- Indicated to salvage joint function in mature dogs, with severe osteoarthritis that is unresponsive to medical therapy
- Pain-free joint function—reported in more than 90% of cases
- Hip joint replacement in only one leg provides acceptable function in approximately 80% of cases
- Complications—dislocation (luxation); damage to the sciatic nerve; infection

Excision Arthroplasty (surgical removal of the "ball" part of the hip joint)

- Removal of the "ball" (femoral head and neck) to eliminate joint pain; the muscles "act" as the joint
- Primarily a salvage procedure—for significant osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage)—when pain cannot be controlled medically or when total hip replacement is cost-prohibitive

- Best results—small, light dogs (weighing less than 20 kg or 44 lbs); patients with good hip musculature
- Pain free function, even in giant-breed dogs
- Slightly abnormal gait often persists following surgery
- Postoperative loss of muscle mass (muscle atrophy) in the hind limbs—common, particularly in large dogs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Pain relieving drugs (known as “analgesics”) and anti-inflammatory drugs—minimize joint pain (and thus stiffness and loss of muscle mass [muscle atrophy] caused by limited usage); decrease inflammation of the lining of the joint (known as “synovitis”); drugs that relieve pain and decrease inflammation include carprofen; etodolac; deracoxib
- Medical therapy—does not correct the structural or biomechanical abnormality; deterioration or degeneration of the hip joint likely to progress; medical therapy often provides only temporary relief of signs
- Glucosamine and chondroitin sulfate—may have a cartilage protective effect in osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage)



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor signs, degree of lameness, and changes seen on X-rays—assess progression
- Medical treatment—if poor response or initial response is followed by deterioration of condition, change the dosage of medication or try a different medication or consider surgical intervention
- Triple pelvic osteotomy—monitored by X-rays, taken periodically; assess healing, metal-implant stability, reestablishment of corresponding surfaces between the “ball” (femoral head) and the “socket” (acetabulum) making up the hip joint (that is, joint congruence), and progression of osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage)
- Hip replacement—monitored by X-rays; assess metal implant stability

PREVENTIONS AND AVOIDANCE

- Best prevented by not breeding dogs affected with hip dysplasia
- Pelvic X-rays—may help identify dogs with actual bony changes of hip dysplasia; may not identify all dogs carrying the disease
- Do not repeat dam–sire breeding that result in affected offspring
- Special diets designed for rapidly growing large-breed dogs—may decrease severity of hip dysplasia

EXPECTED COURSE AND PROGNOSIS

- Joint deterioration or degeneration usually progresses—most patients lead normal lives with proper medical or surgical management



KEY POINTS

- Hip dysplasia has a genetic (inherited) basis, involving multiple genes
- Development of hip dysplasia determined by an interaction of genetic and environmental factors
- Medical therapy is designed to relieve signs (known as “palliative therapy”); it does not “cure” the disease, because the joint instability is not corrected
- Joint deterioration or degeneration often progresses, unless a corrective orthopedic surgical procedure is performed early in the disease
- Surgical procedures can salvage hip-joint function once severe joint deterioration or degeneration occurs

DIFFICULT BIRTH (DYSTOCIA)



BASICS

OVERVIEW

- “Dystocia” is the medical term for difficult birth
- The female dog is a “bitch;” the female cat is a “queen”

GENETICS

- Some forms of difficult birth (dystocia) may be hereditary—inactivity or lack of forceful contractions of the uterus (known as “uterine inertia”), conformational defects in anatomy (pelvic diameter)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

Dogs

- Higher incidence with miniature and small breeds due to small litter size; may occur in large breeds with large litters
- Short-nosed, flat-faced (known as “brachycephalic”) breeds—broad head and narrow pelvis, such as in bulldogs, Boston terriers, pugs
- Large fetal head-to-maternal pelvis ratio—Sealyham terrier, Scottish terrier
- Inactivity or lack of forceful contractions of the uterus (uterine inertia)—Scottish terrier, dachshund, Border terrier, Aberdeen terrier, Labrador retriever
- Miscellaneous dog breeds with overall increased incidence of difficult birth (dystocia)—Chihuahua, dachshund, Pekingese, Yorkshire terrier, miniature poodle, Pomeranian

Cats

- Short-nosed, flat-faced (brachycephalic) breeds—Persian, Himalayan
- Breeds with long head and nose (known as “dolichocephalic breeds”)—Devon rex

Mean Age and Range

- Likelihood of difficult birth (dystocia) increases with age

SIGNS/OBSERVED CHANGES in the ANIMAL

- More than 30 minutes of persistent, strong, abdominal contractions without expulsion of offspring
- More than 4 hours from the onset of strong, abdominal contractions to delivery of first offspring (length of time can vary based on individual animal and species; however, if more than 4 hours have passed without delivery of first offspring, contact your pet’s veterinarian)
- More than 2 hours between delivery of offspring (length of time can vary based on individual animal and species; however, if more than 2 hours have passed between delivery of offspring, contact your pet’s veterinarian)
- Failure of a bitch to go into labor within 24 hours of the drop in rectal temperature below 37.2° C (99° F) or within 36 hours of serum progesterone dropping to less than 2 ng/ml
- Female cries, displays signs of pain, and constantly licks the vulvar area (external genitalia) when delivering
- Prolonged pregnancy or gestation in the bitch—more than 72 days from day of first mating; more than 59 days from the first day following “standing heat” (diestrus) as demonstrated by microscopic examination of vaginal cells (dogs); more than 66 days from luteinizing hormone peak; “luteinizing hormone” is a female hormone—it maintains the “corpus luteum” or “yellow body” in the ovary that produces the female hormone progesterone, which supports and maintains the pregnancy
- Presence of greenish-black discharge preceding the birth of first offspring by more than 2 hours
- Presence of bloody discharge prior to delivery of first offspring or between fetuses

CAUSES

Fetal

- Oversize—singleton litters (that is a litter with only a single fetus); fetal monsters, build-up of fluid (known as “edema”) in the tissues of the fetus (known as “fetal anasarca”); build-up of fluid in specific areas of the brain of the fetus (known as “fetal hydrocephalus”); prolonged pregnancy or gestation due to inability of a singleton fetus to initiate labor
- Abnormal presentation, position, or posture of fetus in the birth canal
- Fetal death

Maternal

- Poor uterine contractions—uterine muscle defect; biochemical imbalance; psychogenic disturbance; exhaustion
- Ineffective abdominal press—pain; fear; debility (exhaustion); diaphragmatic hernia; perforated windpipe (trachea); age
- Inflammation of the placenta (known as “placentitis”), inflammation of the uterus (known as “metritis”), inflammation of the lining of the uterus (known as “endometritis”)
- Pregnancy toxemia, gestational diabetes
- Abnormal pelvic canal from previous pelvic injury, abnormal conformation, or pelvic immaturity
- Congenitally small pelvis—Welsh corgis; short-nosed, flat-faced (brachycephalic) breeds; “congenitally” refers to congenital, which is

something that is present at birth

- Inguinal hernia
- Abnormality of the vaginal vault—narrowing of the birth canal or vagina (known as a “stricture”); presence of a thin wall dividing the birth canal into two canals (known as “vaginal septae”); enlargement or thickening of the wall of the birth canal (known as “vaginal hyperplasia”); vaginal cyst; cancer; poorly developed vagina (known as a “hypoplastic vagina”)
- Abnormality of the vulvar opening—narrowing of the vulva (external genitalia); inverted vulva; small vulva; scarring of the vulva from trauma; cancer
- Insufficient cervical dilation
- Lack of adequate lubrication
- Twisting of the uterus (known as “uterine torsion”)
- Uterine rupture
- Uterine tumors, cysts or scar tissue (known as “adhesions”)

RISK FACTORS

- Age
- Short-nosed, flat-faced (brachycephalic) breeds and toy breeds
- Persian and Himalayan
- Obesity
- Abrupt changes in environment during the period before, during, or after delivery (known as the “peripartum period”)
- Previous history of difficult birth (dystocia)



TREATMENT

HEALTH CARE

- Inpatient—until delivery of all offspring and mother has stabilized
- Fluid replacement—balanced electrolyte solutions; for clinical dehydration
- Inactivity or lack of forceful contractions of the uterus (uterine inertia)—initiate medical treatment, if no evidence of fetal stress; uterine inertia may be due to low blood sugar (known as “hypoglycemia”), low levels of calcium in the blood (known as “hypocalcemia”) or inadequate production of oxytocin or inadequate response to normal oxytocin production; “oxytocin” is a female hormone that causes uterine contractions and promotes milk release during lactation; appropriate treatment should be started once the cause of uterine inertia has been determined
- Medications to stimulate uterine contraction should not be administered in the face of possible obstructive dystocia as they may accelerate placental separation and fetal death, or may cause uterine rupture
- Hypoglycemia—administration of balanced electrolyte solution with 5% to 10% dextrose intravenously
- Hypocalcemia—administration of 10% calcium gluconate by injection
- Whelpwise® monitoring systems can be used to monitor fetal heart rates and uterine contractions and are excellent for bitches with a prior history of uterine inertia or with large litters to determine need for and response to medical treatment

Manual Delivery

- To deliver a fetus lodged in the birth canal
- Apply lubrication liberally; place patient in a standing position
- Digital manipulation—least amount of damage to fetus and dam
- Instrument delivery (dogs)—if the birth canal is too small for digital manipulations
- Use extreme caution; undesirable sequelae include mutilation of the fetus and laceration of the birth canal
- Traction should not be applied to the lower legs of a live fetus
- Cats—use of instruments not recommended because of the small size of the birth canal
- Failure to deliver the fetus within 30 minutes—cesarean section indicated

SURGERY

- Indications for cesarean section—inactivity or lack of forceful contractions of the uterus (uterine inertia) unresponsive to oxytocin or a responsive uterine inertia, but with more than 4 fetuses remaining in the uterus (to maximize fetal survivability); pelvic or vaginal obstruction; inability to correct fetal malposition; fetal oversize; fetal stress; fetal death in the uterus
- Elective cesarean section—breeds prone to difficult birth (dystocia); bitches with a history of dystocia; bitches with a single fetus in the litter or very large litters; elective cesarean section often performed to maximize fetal survivability

Anesthesia

General Comments

- Provide fluids during surgery
- The pregnant uterus can put pressure on the large blood vessels and decrease blood return to the heart, while putting pressure on the diaphragm resulting in decreased volume of air being inspired during a single breath
- Pre-oxygenation of patients will improve maternal and newborn outcome
- A variety of medications can be used during the preoperative, operative, and postoperative periods; they include such medications as glycopyrrolate, atropine, propofol, gas anesthetics (such as isoflurane or sevoflurane), diazepam, ketamine, butorphanol, and oxymorphone; your pet’s veterinarian will choose the anesthetic protocol for your pet



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Oxytocin—to stimulate contractions for cases with inactivity or lack of forceful contractions of the uterus (uterine inertia)



FOLLOW-UP CARE

PATIENT MONITORING

- Ultrasound examination—recommended; monitor fetal heart rate during medical management of inactivity or lack of forceful contractions of the uterus (uterine inertia)

PREVENTIONS AND AVOIDANCE

- Schedule elective cesarean section for bitches with abnormal pelvic canal; small pelvis; birth canal abnormalities; breeds likely to have difficult birth (dystocia); dams with previous history of inactivity or lack of forceful contractions of the uterus (uterine inertia)
- Scheduling of surgery should be based on timing of ovulation and breeding to ensure acceptable fetal survivability

POSSIBLE COMPLICATIONS

- Increased risk of difficult birth (dystocia) in future pregnancies with some causes
- Loss of puppies or kittens in the litter, if treatment is not begun promptly

EXPECTED COURSE AND PROGNOSIS

- If difficult birth (dystocia) is identified promptly and intervention is successful—prognosis is good to fair for life of the dam; fair for survival of offspring
- If dystocia unrecognized or untreated for 24 to 48 hours—prognosis is poor to guarded for life of the dam; unlikely that any offspring will survive (dogs); highly variable depending on cause (cats)



KEY POINTS

- “Dystocia” is the medical term for difficult birth
- Schedule elective cesarean section for bitches with abnormal pelvic canal; small pelvis; birth canal abnormalities; breeds likely to have difficult birth (dystocia); dams with previous history of inactivity or lack of forceful contractions of the uterus (uterine inertia)
- Identify difficult birth (dystocia) quickly and when it occurs, contact your pet’s veterinarian for appropriate treatment
- If difficult birth (dystocia) is identified promptly and intervention is successful—prognosis is good to fair for life of the dam; fair for survival of offspring
- If dystocia unrecognized or untreated for 24 to 48 hours—prognosis is poor to guarded for life of the dam; unlikely that any offspring will survive (dogs); highly variable depending on cause (cats)

DISEASES CAUSED BY VARIOUS *EHRlichia* (EHRlichiosis)



BASICS

OVERVIEW

- “Ehrlichiosis” is the medical term for diseases caused by various species of *Ehrlichia*
- *Ehrlichia* species are disease-causing agents that are transmitted by ticks
- In addition, two other genera, known as *Anaplasma* and *Neorickettsia*, are included in the same family of organisms as *Ehrlichia* (and some were previously named as *Ehrlichia* species), thus they are included under the diseases known as “ehrlichiosis”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Long-term (chronic) ehrlichiosis, caused by *Ehrlichia canis*—seems more severe in Doberman pinschers and German shepherd dogs

Mean Age and Range

- Average age—5.22 years
- Range—2 months to 14 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Duration of clinical signs from initial sudden (acute) illness to presentation to the pet’s veterinarian is usually greater than 2 months
- Sluggishness (lethargy)
- Depression
- Lack of appetite (known as “anorexia”) and weight loss
- Fever
- Spontaneous bleeding—sneezing, bleeding from the nose or nasal passages (known as “epistaxis” or a “nose bleed”)
- Breathing distress
- Wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)
- Head tilt
- Eye pain (inflammation of the iris [colored part of the eye] and other areas in the front part of the eye [known as “uveitis”])

Sudden (Acute) Ehrlichiosis

- Bleeding disorder, characterized by pinpoint areas of bleeding (known as “petechia”) in the gums, as a result of a low platelet count (known as “thrombocytopenia;” “platelets” are normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding;)
- Fever, depression, lack of appetite (anorexia), weight loss
- Generalized enlarged lymph nodes (known as “lymphadenopathy”)
- Ticks—found in 40% of cases
- Difficulty breathing (known as “dyspnea”) and possible bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); increased lung sounds may be noted when listening to the chest with a stethoscope (known as “auscultation”)
- Widespread (diffuse) central nervous system disease (inflammation of the membranes covering the brain and spinal cord [known as “meningitis”])
- Wobbly, incoordinated or “drunken” appearing gait or movement (ataxia)
- The animal’s sense of balance is altered (known as a “vestibular disorder”)
- Generalized or localized areas where the animal is overly sensitive to pain or touch (known as “hyperesthesia”)
- Most dogs recover without treatment and enter a subclinical state; a “subclinical state” is one in which the animal is infected, but has no signs of disease

Long-Term (Chronic) Ehrlichiosis

- Spontaneous bleeding
- Low red-blood cell count (known as “anemia”)
- Generalized enlarged lymph nodes (lymphadenopathy)

- Fluid build-up (known as “edema”) in the scrotum and legs
- Enlarged spleen (known as “splenomegaly”)
- Enlarged liver (known as “hepatomegaly”)
- Inflammation of the iris (colored part of the eye) and other areas in the front part of the eye (uveitis)
- Blood in the anterior chamber of the eye (the front part of the eye, between the cornea and the iris; accumulation of blood known as “hyphema”)
- Bleeding into the back of the eye (known as “retinal hemorrhages”) and separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid;” condition known as “retinal detachment”) with blindness
- Fluid build-up in the clear part of the eye (known as “corneal edema”)
- Inflammation of the joints (known as “arthritis”)—rare
- Seizures—rare

CAUSES

- Dogs—can be infected with a number of species of *Ehrlichia*; *Ehrlichia canis*, *Anaplasma platys* (formerly *Ehrlichia platys*), *Anaplasma phagocytophila* (formerly *Ehrlichia equi* and *Ehrlichia phagocytophila*), *Ehrlichia ewingii*, and *Ehrlichia chaffeensis* produce main disease entities
- Cats—*Neorickettsia risticii* (formerly *Ehrlichia risticii*); possibly a species similar to *Ehrlichia canis*

RISK FACTORS

- Coexistent infection with other blood parasites (such as *Babesia*, *Haemobartonella*, *Anaplasma platys*, and *Hepatozoon canis*)—worsens clinical syndrome



TREATMENT

HEALTH CARE

- Inpatient—initial medical stabilization for low red-blood cell count (anemia) and/or bleeding tendency resulting from low platelet count (thrombocytopenia)
- Outpatient—stable patients; monitor blood and response to medication frequently
- Balanced fluids are indicated for dehydration
- Blood transfusion may be indicated for low red-blood cell count (anemia)
- Platelet-rich plasma or a blood transfusion is indicated for bleeding resulting from a low platelet count (thrombocytopenia)

ACTIVITY

- Restricted

SURGERY

- If surgery is needed for other reasons, blood transfusion may be needed to correct low red-blood cell count (anemia) and/or low platelet count (thrombocytopenia)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Doxycycline—generally administered by mouth for 14 to 28 days; may be given intravenously, if the dog is vomiting
- Imidocarb dipropionate—effective against both *Ehrlichia canis* and *Babesia*; reasonable alternative to doxycycline
- Steroids—prednisolone or prednisone; may be indicated when the low platelet count (thrombocytopenia) is life-threatening (thrombocytopenia thought to be a result of immune-mediated mechanisms); because immune-mediated thrombocytopenia unrelated to ehrlichiosis is a principal differential diagnosis, steroids may be indicated until results of serologic tests (blood tests that detect the presence of antibodies to a certain disease-causing agent or antigen; an “antibody” is a protein that is produced by the immune system in response to a specific antigen) are available
- Androgenic steroids—to stimulate bone-marrow production of blood cells in dogs that have been infected for a prolonged time and that

have decreased ability to produce blood cells in their bone marrow; androgenic steroids include oxymetholone and nandrolone

- Other antibiotics that may be administered include oxytetracycline or tetracycline (effective and less expensive) or chloramphenicol (avoids yellow discoloration of erupting teeth caused by tetracyclines in puppies under 6 months of age; however, chloramphenicol does have potentially serious side effects; discuss the use of chloramphenicol with your pet's veterinarian)



FOLLOW-UP CARE

PATIENT MONITORING

- Platelet count—every 3 days after initiating treatment, until normal; improvement is rapid in sudden (acute) infection
- Serologic testing (blood tests that detect the presence of antibodies to a certain disease-causing agent or antigen; an “antibody” is a protein that is produced by the immune system in response to a specific antigen)—repeat in 9 months; most dogs will become negative on the testing (known as “seronegative status”); positive titer suggests reinfection (prior infection does not imply protective immunity) or ineffective treatment (repeat treatment regimen)

PREVENTIONS AND AVOIDANCE

- Control tick infestation—use products to kill and/or repel ticks as directed by your pet's veterinarian; any such products must be used according to the package label; flea and tick collars may reduce reinfestation, but reliability has not been proven; avoid tick-infested areas
- Removing ticks by hand—use gloves; ensure mouth parts are removed to avoid a foreign-body reaction

EXPECTED COURSE AND PROGNOSIS

- Sudden (acute) ehrlichiosis—excellent prognosis with appropriate treatment
- Long-term (chronic) ehrlichiosis—may take 4 weeks for a clinical response; prognosis poor with decreased ability to produce blood cells in the bone marrow (known as “hypoplastic marrow”)



KEY POINTS

- Sudden (acute) ehrlichiosis—prognosis excellent with appropriate therapy
- Long-term (chronic) ehrlichiosis—response may take 4 weeks; prognosis poor with severely decreased ability to produce blood cells in the bone marrow (severe hypoplastic marrow)
- Progression from sudden (acute) to long-term (chronic) ehrlichiosis can be prevented easily by early, effective treatment; but many dogs remain positive on serologic tests (blood tests that detect the presence of antibodies to a certain disease-causing agent or antigen; an “antibody” is a protein that is produced by the immune system in response to a specific antigen) and may relapse (even years later)
- Doberman pinschers and German shepherd dogs appear to have a more long-term (chronic) and severe form of disease than other breeds

ENCEPHALITIS (INFLAMMATION OF THE BRAIN)



BASICS

OVERVIEW

- “Encephalitis” is the medical term for inflammation of the brain
- May be accompanied by inflammation of the spinal cord (known as “myelitis”) and/or meninges (the membranes covering the brain and spinal cord; inflammation of the meninges known as “meningitis”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Inflammation of the brain and the membranes covering the brain (meninges) characterized by nodular, inflammatory lesions (known as “granulomatous meningoencephalitis”)—mostly small-breed dogs, especially terriers and miniature poodles; large-breed dogs also affected
- Inflammation of the brain (known as “pug encephalitis”)—pugs
- Inflammation of the brain, spinal cord, and membranes covering them with mononuclear cells and neutrophils (pus) present in the tissues (known as “pyogranulomatous meningoencephalitis”)—German shorthaired pointers
- Inflammation of the brain (known as “Maltese encephalitis”)—Maltese
- Inflammation of the brain characterized by the destruction and death of nervous tissue (known as “Yorkshire terrier necrotizing encephalitis”)—Yorkshire terriers

SIGNS/OBSERVED CHANGES in the ANIMAL

- Usually a very sudden (peracute) to sudden (acute) onset of clinical signs that rapidly progress
- Inflammation of the brain and the membranes covering the brain (meninges) characterized by nodular, inflammatory lesions (granulomatous meningoencephalitis) and fungal or protozoal caused inflammation of the brain (encephalitis)—sometimes signs progress over a more long-term (chronic) course
- Fever, lung disease, and/or gastrointestinal disturbances—usually precede inflammation of the brain (encephalitis)
- With mycotic, rickettsial, viral, and protothecal organisms—lesions in the retina (back part of the eye) are frequent
- Signs are determined by the portion of the brain most affected; front part of the brain—seizures; circling; pacing; personality change; decreasing level of responsiveness; back part of the brain—abnormalities related to the brainstem (drowsiness or sleepiness [known as “somnia”]), head tilt, weakness or partial paralysis to paralysis of the facial muscles, incoordination
- Progression (such as unequal size of the pupils [known as “anisocoria”], pinpoint pupils, decreasing level of consciousness, short, rapid movements of the eyeball [known as “nystagmus”])—suggests the brain has pushed downward in the skull and has herniated through the opening that leads to the neck (known as “tentorial herniation”)

CAUSES

Dogs

- Unknown cause (so called “idiopathic” disease) or immune-mediated disease—inflammation of the brain and the membranes covering the brain (meninges) characterized by nodular, inflammatory lesions (granulomatous meningoencephalitis); pug encephalitis; Maltese encephalitis; Yorkshire terrier necrotizing encephalitis; inflammation of the brain and membranes covering the brain (meninges), characterized by the presence of a type of white-blood cell, the eosinophil (condition known as “eosinophilic meningoencephalitis”)
- Viral infections—canine distemper virus; rabies virus; herpes virus; parvovirus; adenovirus; pseudorabies; Eastern and Venezuelan equine encephalomyelitis virus
- Inflammation of the brain and spinal cord following vaccination (known as “postvaccinal encephalomyelitis”)—canine distemper virus; rabies virus; canine coronavirus-parvovirus
- Rickettsial disease—Rocky Mountain spotted fever; ehrlichiosis
- Fungal or mycotic disease—cryptococcosis; blastomycosis; histoplasmosis; coccidioidomycosis; aspergillosis; phaeohyphomycosis
- Bacterial infections
- Protozoal disease—toxoplasmosis; neosporosis; encephalitozoonosis
- Spirochetes—borreliosis (Lyme disease)
- Parasite migration—heartworms (*Dirofilaria immitis*); roundworms (*Toxocara canis*); hookworms (*Ancylostoma caninum*); *Cuterebra*; cysticercosis (where a tapeworm larva has embedded itself in nervous tissue)

- Migrating foreign body—plant awn; others
- Protothecosis (infection with a type of algae, *Prototheca*)

Cats

- Unknown cause (so called “idiopathic” disease) or immune-mediated disease—inflammation of the brain and the membranes covering the brain (meninges) characterized by nodular, inflammatory lesions (granulomatous meningoencephalitis); inflammation of the brain and membranes covering the brain (meninges), characterized by the presence of a type of white-blood cell, the eosinophil (eosinophilic meningoencephalitis)
- Inflammation of the gray matter of the brain and spinal cord for unknown cause (condition known as “idiopathic polioencephalomyelitis”)
- Viral disease—feline infectious peritonitis (FIP); rabies; feline immunodeficiency virus (FIV); pseudorabies; panleukopenia; rhinotracheitis
- Fungal or mycotic disease—cryptococcosis; blastomycosis; phaeohyphomycoses
- Bacterial infections
- Protozoal disease—toxoplasmosis
- Parasite migration—heartworms (*Dirofilaria immitis*); *Cuterebra*

RISK FACTORS

- Medications that decrease the immune response (known as “immunosuppressive drugs”) and feline immunodeficiency virus (FIV) or feline leukemia virus (FeLV) infection—infectious causes of inflammation of the brain (encephalitis)
- Tick-infected areas—rickettsial and *Borrelia* infections
- Travel history—fungal or mycotic infections



TREATMENT

HEALTH CARE

- Inpatient—diagnosis and initial therapy
- Symptomatic treatment—control fluid build-up in the brain (known as “brain edema”) and seizure activity, as necessary
- Fluid build-up in the brain (brain edema)—20% mannitol; may repeat within 1 to 2 hours to achieve maximum response; limit fluids to prevent rebound fluid build-up; with mannitol, short-term steroid treatment (dexamethasone) may be indicated for further control
- Seizures—treat with medications to control seizures (known as “antiepileptic drugs”); control may be erratic until the inflammation of the brain (encephalitis) is treated

ACTIVITY

- As tolerated

DIET

- With severe depression or vomiting—nothing by mouth until condition improves, to prevent aspiration

SURGERY

- Brain biopsy—may be needed for specific diagnosis



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Apply specific therapy once diagnosis is reached or highly suspected
- Unknown cause (so called “idiopathic” disease) or immune-mediated disease—steroids (prednisone), as directed by your pet’s veterinarian
- Rickettsial disease and borreliosis (Lyme disease)—doxycycline
- Protozoal disease—clindamycin

- Fungal or mycotic—requires treatment for 1 to 2 years; use itraconazole or fluconazole; steroids often are needed during the first 4 to 6 weeks to control fluid build-up in the brain (brain edema)
- Viral and postvaccinal inflammation of the brain (encephalitis)—no definitive treatment; treat symptomatically
- Bacterial disease—broad-spectrum antibiotics that penetrate the blood–brain barrier; if bacteria is not identified as to actual type, a combination of enrofloxacin and ticarcillin-clavulanate or amoxicillin-clavulanate may be tried
- CCNU (lomustine)—treat dogs for one year; may be effective in causing remission in dogs with immune-mediated inflammation of the brain (encephalitis) that does not respond to prednisone; adverse effects include bone-marrow suppression, leading to low red-blood cell and low white-blood cell counts; gastrointestinal effects; and liver toxicity
- Cyclosporine modified—can be used with prednisone, CCNU and cytarabine for 1.5 years to achieve maximum decrease in the immune response (immune suppression) in dogs with immune-mediated inflammation of the brain (encephalitis)
- Cytarabine—after CCNU in dogs with immune-mediated inflammation of the brain (encephalitis) to achieve further decrease in the immune response (immune suppression); adverse effects are similar to those of CCNU, but much milder than side effects seen with CCNU



FOLLOW-UP CARE

PATIENT MONITORING

- Frequent nervous system evaluations in the first 48 to 72 hours to monitor progress
- Relapse as medication is withdrawn—repeat cerebrospinal fluid (CSF) analysis
- Measure serum titer of *Cryptococcus* capsular antigen every 3 months until negative, if cryptococcosis is diagnosed
- If treated with CCNU (lomustine), a complete blood count (CBC) should be done at 1 and 3 weeks following treatment; serum chemistry profile and serum bile acids should be done at 3 weeks and then every 2 months
- If treated with cytarabine, a complete blood count (CBC) should be done 2 weeks following treatment

PREVENTIONS AND AVOIDANCE

- A method of effective tick control should be used on animals that live in an area where ticks are common
- Avoid vaccination of dogs that have had inflammation of the brain and the membranes covering the brain (meninges) characterized by nodular, inflammatory lesions (granulomatous meningoencephalitis)

POSSIBLE COMPLICATIONS

- Long-term steroid therapy—signs of excessive levels of steroids in the blood following treatment (known as “iatrogenic hyperadrenocorticism” or “iatrogenic Cushing’s disease”)
- Cerebrospinal fluid (CSF) collection or natural course of the disease—brain may push downward in the skull and herniate through the opening that leads to the neck (tentorial herniation)
- Death

EXPECTED COURSE AND PROGNOSIS

- Inflammation of the brain (encephalitis) is life-threatening, if left untreated
- Resolution of signs—generally gradual (2 to 8 weeks)
- Protothecal (algae) inflammation of the brain (encephalitis)—almost always progresses to death
- Immune-mediated disease—fair to good prognosis for complete remission with aggressive treatment to decrease the immune response (immunosuppression)
- Rickettsial, fungal or mycotic, bacterial, protozoal, and spirochete infections—fair chance of survival
- Parasite migration, migrating foreign bodies, pyogranulomatous meningoencephalitis, Yorkshire terrier necrotizing encephalitis, and polioencephalomyelitis—usually fatal
- Pug and Maltese encephalitis—may be fatal; course varies greatly; some patients respond to steroid treatment for long periods
- Inflammation of the brain and spinal cord following vaccination (postvaccinal encephalomyelitis)—may resolve on its own; often permanent damage and death



KEY POINTS

- Inflammation of the brain (encephalitis) is life-threatening, if left untreated
- Relapse is possible with idiopathic (of unknown cause) or immune-mediated inflammation of the brain (encephalitis) when therapy is discontinued

ATRIOVENTRICULAR VALVE ENDOCARDIOSIS



BASICS

OVERVIEW

- “Atrioventricular valve” refers to the heart valves between the top chamber (known as the “atrium”) and the bottom chamber (known as the “ventricle”) of the heart; two atrioventricular valves are present in the heart—one on the right side of the heart and one on the left side of the heart
- “Endocardiosis” is the medical term for long-term (chronic) formation of excessive fibrous tissue of the atrioventricular valves
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- The atrioventricular valves are the tricuspid valve (right side) and the mitral valve (left side)
- “Atrioventricular valve endocardiosis” is a long-term (chronic) disease characterized by a decline in the function or structure of the tricuspid and/or mitral valves, leading to inability of the valves to work properly (known as “valvular insufficiency”) and congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

GENETICS

- Not established

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Mainly dogs, but may be seen in old cats

Breed Predilections

- Typically small breeds
- Highest prevalence—Cavalier King Charles spaniel, Chihuahua, miniature schnauzer, Maltese, Pomeranian, cocker spaniel, Pekingese, fox terrier, Boston terrier, miniature poodle, toy poodle, miniature pinscher, and whippet

Mean Age and Range

- Onset of congestive heart failure at 10 to 12 years of age, although may detect a murmur several years earlier; Cavalier King Charles spaniels typically affected much earlier (6 to 8 years of age); “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

Predominant Sex

- Males are 1.5 times more likely to have atrioventricular valve endocardiosis than are females

SIGNS/OBSERVED CHANGES in the ANIMAL

Asymptomatic Valve Disease (pet has no clinical signs of heart-valve disease)

- Heart murmur
- As the disease progresses, the heart murmur typically gets louder and radiates more widely; with severe disease, the murmur may decrease in frequency and loudness

Mild Congestive Heart Failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)

- Coughing; exercise intolerance; and difficulty breathing (known as “dyspnea”) with exercise

Moderate Congestive Heart Failure

- Coughing; exercise intolerance; and difficulty breathing (dyspnea) at all times

Severe Congestive Heart Failure

- Severe difficulty breathing (dyspnea); profound weakness; abdominal swelling or distention; productive coughing (that is, coughing up pink, frothy fluid); standing with the elbows away from the body in an attempt to increase lung capacity (known as “orthopnea”); bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red-blood cells (condition known as “cyanosis”); and fainting (known as “syncope”)

CAUSES

- Unknown (so called “idiopathic disease”)



TREATMENT

HEALTH CARE

- Treat patients that need oxygen support as inpatients; if stable, patients may be treated at home, where they may be less stressed
- Oxygen therapy as needed for low levels of oxygen in the blood (known as “hypoxemia”)

ACTIVITY

- Absolute exercise restriction for patients with clinical signs
- Stable patients receiving medical treatment—restrict exercise to leash walking; avoid sudden, intense exercise

DIET

- A salt-restricted diet is recommended, if tolerated, for a pet in congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Low levels of sodium in the blood (known as “hyponatremia”) may develop as congestive heart failure progresses and in patients fed severely salt (sodium)—restricted diets in conjunction with medications to remove excess fluid from the body (known as “diuretics”) and certain heart medications (angiotensin-converting enzyme [ACE] inhibitors)
- If the pet develops low levels of sodium in the blood (hyponatremia), switch to a less salt (sodium)-restricted diet (such as kidney or geriatric diet)

SURGERY

- Surgical heart valve replacement and purse-string suture techniques to reduce the area of the opening of the mitral valve have been used; experience with these techniques is limited, but surgical repair may be an option when access to a cardiovascular surgeon and heart-lung bypass (known as “cardiopulmonary bypass”) are available



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Recommended treatment depends on stage of the disease

Asymptomatic Patients (pet has no clinical signs of heart-valve disease)

- No treatment may be needed, if patient has no indication of heart enlargement identified through diagnostic tests

Mild or Moderate Congestive Heart Failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)

- Medications to remove excess fluid from the body (diuretics)—furosemide
- Heart medications, such as ACE inhibitors (examples are enalapril and benazepril), digoxin, calcium channel blockers, β -blockers, nitroglycerin ointment
- Spironolactone, while typically used for its diuretic effect in combination with other diuretics, has been shown to have a positive influence as heart disease progresses

Severe Congestive Heart Failure

- Oxygen—administered in an oxygen cage or through a nasal catheter
- Medications to remove excess fluid from the body (diuretics)—furosemide

Medications to Dilate or Enlarge Blood Vessels (Known as “Vasodilators”)

- Enalapril or benazepril
- Hydralazine
- Nitroglycerin ointment or injectable
- Sodium nitroprusside
- Pimobendan used alone or in combination with other vasodilators and/or digoxin

Medications that Improve Heart-Muscle Contraction (Known as “Positive Inotropes”)

- Digoxin
- Pimobendan
- Dobutamine
- Dopamine
- β -blockers (such as carvedilol)



FOLLOW-UP CARE

PATIENT MONITORING

- Take a baseline chest X-ray when a heart murmur is first detected and every 6 to 12 months thereafter to document progressive enlargement of the heart
- After an episode of congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs), check pet weekly during the first month of treatment; may repeat chest X-rays and an electrocardiogram (“ECG,” a recording

of the electrical activity of the heart) at the first weekly checkup and on subsequent visits, if any changes are seen on physical examination

- Monitor blood work (blood urea nitrogen [BUN] and creatinine) when medications to remove excess fluid from the body (diuretics) and ACE inhibitors are used in combination
- Monitor serum potassium levels when spironolactone (another diuretic) and ACE inhibitors are used together, especially when combined with digoxin

POSSIBLE COMPLICATIONS

- Inflammation of the lining of the heart (known as “endocarditis”) because of bacteria infecting the diseased mitral valve

EXPECTED COURSE AND PROGNOSIS

- Progressive deterioration (degeneration) of valve changes and heart-muscle function occurs, necessitating increasing drug dosages
- Long-term prognosis depends on response to treatment and stage of congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)



KEY POINTS

- Atrioventricular valve endocardiosis is a progressive disease
- It is important to dose all medications consistently and to provide diet and exercise management
- If the pet develops worsening clinical signs or experiences unexpected changes in condition, notify the veterinarian immediately

INFLAMMATION/INFECTION OF THE LINING OF THE HEART, USUALLY INVOLVING THE HEART VALVES (INFECTIVE ENDOCARDITIS)



BASICS

OVERVIEW

- Inflammation and infection of the lining of the heart, usually involving the heart valves
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- Infectious agents usually are gram-positive bacteria, especially staphylococci or streptococci; occasionally *Rickettsia* or *Bartonella* in dogs
- Infectious agents rarely are fungi in dogs; cases that have negative bacterial culture results may have infection by *Bartonella* or *Aspergillus*, a type of fungus; less likely is infection by *Brucella*, *Coxiella*, and *Chlamydia*

GENETICS

- Genetic susceptibility is unlikely

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs; rarely cats

Breed Predilection

- Middle-sized to large breeds
- Breeds susceptible to a birth defect involving narrowing just below the aortic valve, the heart valve from the left ventricle to the aorta (the main artery of the body; condition known as “subaortic stenosis”)

Mean Age and Range

- Most affected dogs are 4 to 6 years of age; infection can occur at any age

Predominant Sex

- Most studies report males are more likely to be affected than females—may be as great as a 2:1 ratio of males-to-females

SIGNS/OBSERVED CHANGES in the ANIMAL

- The presence of gram-negative bacteria in the blood (known as “gram-negative bacteremia”) results in very sudden (known as “peracute”) or sudden (known as “acute”) clinical signs; the presence of gram-positive bacteria in the blood (known as “gram-positive bacteremia”) results in signs over a moderate amount of time (known as “subacute”) or long-term (known as “chronic”)
- Generalized (systemic) signs are secondary to sudden lack of blood supply that leads to death of tissues (known as “infarction”), infection, or damage due to the body’s immune response; usually override heart signs (such as congestive heart failure [CHF] and/or irregular heart beats [arrhythmias]); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs—CHF signs include cough; difficulty breathing (known as “dyspnea”); bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”)
- May have had signs of infectious disease involving the mouth, gastrointestinal tract, and/or genital tract (such as inflammation/infection of the prostate [prostatitis]) within the past few weeks to several months in some patients
- Animal may have signs of other diseases and/or history of factors that increase the likelihood of infection of the lining of the heart, such as being on medications to decrease the immune response (known as “immunosuppressive drug therapy”), narrowing of the aortic valve, the heart valve from the left ventricle to the aorta (the main artery of the body; condition known as “aortic stenosis”), recent surgery, infected wounds, abscesses, or skin infection characterized by the presence of pus (known as “pyoderma”)
- Sluggishness (lethargy)
- Weakness and/or lameness
- Fever
- Lack of appetite (known as “anorexia”)
- Stomach and/or intestinal disturbances
- General signs of discomfort and “not feeling well” (known as “malaise”)
- Difficulty breathing (dyspnea) caused by congestive heart failure (CHF); congestive heart failure is a condition in which the heart

cannot pump an adequate volume of blood to meet the body's needs

- Irregular heart beats (arrhythmias)
- Single or shifting-leg lameness; “shifting-leg” lameness is characterized by lameness in one leg, then that leg appears to be normal and another leg is involved
- Heart murmur

CAUSES

- Bacterial infection associated with the mouth, bone, prostate, skin, and other sites
- Diagnostic or surgical procedures that may lead to introduction of bacteria into the bloodstream

RISK FACTORS

- Congenital (present at birth) heart defect involving narrowing just below the aortic valve, the heart valve from the left ventricle to the aorta (the main artery of the body; condition is “subaortic stenosis”)
- Decreased ability to produce a normal immune response (known as “immunosuppression”) from treatment with long-term or high-dose steroids or secondary to cancer or administration of chemotherapy



TREATMENT

HEALTH CARE

- Virtually all animals with suspected inflammation/infection of the lining of the heart, usually involving the heart valves (infective endocarditis) should be hospitalized
- Good hydration for patients with generalized bacterial infection (known as “septic patients”), particularly those receiving a particular class of antibiotics (known as an “aminoglycoside”)
- Aggressive fluid therapy—for patients with kidney failure
- Actual or impending (that is, about to occur) congestive heart failure (CHF) limits fluid volumes that can be administered; this problem is virtually insurmountable in patients with coexistent kidney failure
- Impending (that is, about to occur) congestive heart failure (CHF)—cautious use of fluid therapy

ACTIVITY

- Variable—depends on whether or not congestive heart failure (CHF) is present or impending (that is, about to occur)

DIET

- Moderate sodium restriction if congestive heart failure (CHF) is present or impending (that is, about to occur)

SURGERY

- Inflammation/infection of the aortic valve (aortic valve endocarditis)—almost always results in left-sided congestive heart failure (CHF) that is difficult to control medically; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs; surgery to replace the aortic valve is indicated—this surgical procedure routinely is performed in human medicine, but rarely attempted in veterinary medicine because of lack of expertise, lack of facilities, and high cost



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treatment is variable—depends on severity of generalized bacterial infection (sepsis) and presence or absence of congestive heart failure (CHF); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs

Antibiotics

- Backbone of treatment, but usually don't eradicate infection before irreversible heart-valve damage occurs; more than minimal damage to the aortic valve is life-threatening
- High-dose, intravenous (IV) administration of antibiotics designed to kill bacteria (known as “bactericidal antibiotics”) is imperative and recommended for as long as feasible, followed by subcutaneous (SC or under the skin) administration

- Antibiotics administered by mouth—recommended only after at least 4 weeks of injectable therapy and at least 1 week after blood work changes and clinical signs of inflammation and infection have disappeared; long-term (greater than 4 months) treatment required to eradicate the infection from abnormal or damaged heart valves
- Antibiotic selection determined by both the urgency of complications of the generalized bacterial infection (sepsis) and results of bacterial culture; coagulase-positive staphylococci and streptococci are incriminated most often as causing the disease, so choices of antibiotics can be made logically before bacterial culture results are obtained
- Coagulase-positive staphylococci—usually resistant to penicillin and ampicillin
- Streptococci—often resistant to aminoglycosides and fluoroquinolones
- Gram-negative bacteria—often sensitive to third-generation cephalosporins, fluoroquinolones, and aminoglycosides
- *Bartonella*—only aminoglycosides appear bactericidal; can try doxycycline, fluoroquinolone, rifampin, or azithromycin
- First-generation cephalosporins—reasonable choice for stable patients until bacterial culture results are obtained
- Treat life-threatening, generalized bacterial infection (sepsis) immediately with drug combinations; pending bacterial culture results, one of three regimens is recommended: (1) penicillin, ampicillin, ticarcillin, or a first-generation cephalosporin combined with an aminoglycoside—high doses of aminoglycosides cannot be administered, and fluid support with monitoring for kidney toxicity (side effect of aminoglycosides) is required; thus aminoglycosides are not good choices for animals with overt or impending (that is, about to occur) congestive heart failure (CHF) or those with kidney failure; (2) clindamycin plus enrofloxacin; (3) advanced-generation cephalosporins or ticarcillin-clavulanic acid (Timentin®)—high dosages, but only normal dosages if patient has kidney failure

Treatment of Congestive Heart Failure (CHF)

- Various heart medications (such as pimobendan, angiotensin-converting enzyme [ACE] inhibitors, and amlodipine) and medications to remove excess fluid from the body (known as “diuretics,” such as furosemide) are indicated for patients with long-term (chronic) congestive heart failure (CHF); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Oxygen, nitroglycerin, high-dose furosemide, and hydralazine for patients with sudden (acute), severe fluid build-up in the lungs (known as “pulmonary edema”)
- “Blood thinners” or medications to prevent the development of blood clots (known as “anticoagulant therapy”) may be used
- Aspirin and/or dalteparin may reduce the spread of the bacteria and prevent the development of blood clots
- Heparin can be used in the hospital setting to decrease the likelihood of blood-clot formation



FOLLOW-UP CARE

PATIENT MONITORING

- Emergence of antibiotic resistance—relapsing fever and changes in the white-blood cell count indicating inflammation; imperative to adjust treatment on the basis of bacterial culture results
- Weekly physical examination and complete blood count (CBC) after discharge
- Repeat blood cultures 1 week after antibiotics are discontinued or if fever recurs

PREVENTIONS AND AVOIDANCE

- Indwelling catheters—restrict to appropriate usage; place intravenous (IV) catheter into vein using sterile technique (known as “aseptic placement”); replace IV catheter within 3 to 5 days
- Administer antibiotics to animals undergoing dentistry—controversial, except in animals with congenital (present at birth) heart defects and infections of the mouth
- Avoid careless use of steroids

POSSIBLE COMPLICATIONS

- Congestive heart failure (CHF), condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Kidney failure
- Presence of infected blood clots in many tissues and organs
- Persistent or dormant diseases of several joints caused by an immune response (known as “immune-mediated polyarthropathy”)

EXPECTED COURSE AND PROGNOSIS

- Best prognosis associated with short history of bacteria in the blood (bacteremia), rapid diagnosis, and aggressive treatment
- Death rate relatively higher in animals recently given steroids
- Grave prognosis for most patients with inflammation/infection of the aortic valve (aortic valve endocarditis); the aortic valve is the valve from the left ventricle to the aorta (the main artery of the body)

- Dormant congestive heart failure (CHF) may develop months to years later with inflammation/infection of the mitral valve (mitral valve endocarditis); the mitral valve is the heart valve between the left atrium and the left ventricle



KEY POINTS

- Inflammation and infection of the lining of the heart, usually involving the heart valves
- Grave prognosis if the aortic valve is involved; the aortic valve is the valve from the left ventricle to the aorta (the main artery of the body)
- Guarded prognosis if only the mitral valve is involved; the mitral valve is the heart valve between the left atrium and the left ventricle

PROTEIN-LOSING ENTEROPATHY

(DISEASES CAUSING PROTEIN LOSS INTO THE INTESTINAL TRACT)



BASICS

OVERVIEW

- “Enteropathy” is an intestinal disease
- “Protein-losing enteropathy” is any disease process that is characterized by excessive loss of proteins from the body into the gastrointestinal tract; the “gastrointestinal tract” includes the stomach, small intestines, and large intestines
- Diseases associated with protein-losing enteropathy include primary gastrointestinal disease and generalized (systemic) disorders, such as lymphatic disease (“lymphatic” refers to vessels within the body that transports lymph, a clear to slightly colored liquid that contains white-blood cells—it serves many functions including removing bacteria from tissues and it also transports fat from the small intestines; it eventually empties into the blood, returning tissue fluids into the general body circulation) or congestive heart failure (a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)
- Also known as “PLE”

GENETICS

- The hereditary nature of protein-losing enteropathy (PLE) in general or of specific underlying causes of PLE is suspected, based on an increased number of cases in specific dog breeds; however, no genetic basis has been proven so far

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predisposition

- Breeds of dogs with an increased likelihood of developing protein-losing enteropathy compared to other breeds include the soft-coated wheaten terrier, basenji, Yorkshire terrier, and Norwegian lundehund
- Soft-coated wheaten terriers may have protein-losing nephropathy (condition in which proteins are lost from the body through the kidneys) in conjunction with protein-losing enteropathy (PLE)

Mean Age and Range

- Any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs are variable
- Diarrhea (long-term [chronic], continuous or intermittent, watery to semisolid), weight loss, and sluggishness (lethargy) are reported most frequently; however, a significant number of dogs with protein-losing enteropathy (PLE) have normal bowel movements
- Vomiting is reported uncommonly
- Fluid build-up in the abdomen (known as “ascites”); fluid build-up under the skin of the lower part of the body and the legs (known as “dependent edema”); and difficulty breathing (known as “dyspnea”) from fluid build-up in the space between the chest wall and the lungs (known as “pleural effusion”) may be detected with markedly low levels of protein in the blood (known as “marked hypoproteinemia”)
- Thickened loops of intestine may be felt during examination of the abdomen by your pet’s veterinarian

CAUSES

Lymphatic Disorders (“lymphatic” refers to vessels within the body that transports lymph, a clear to slightly colored liquid that contains white-blood cells—it serves many functions including removing bacteria from tissues and returning fluids to the circulation)

- Intestinal lymphangiectasia; “lymphangiectasia” is defined as dilation of the lymphatic vessels in the gastrointestinal tract; the “gastrointestinal tract” includes the stomach, small intestines, and large intestines
- Gastrointestinal lymphoma; “lymphoma” is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body
- Nodular or mass lesions (known as “granulomatous infiltrates”) of the small intestines
- Congestive heart failure leading to increased pressure in the flow of lymph (known as “lymphatic hypertension”); “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs; “lymph” is a clear to slightly colored fluid that contains white-blood cells—it circulates through the lymphatic vessels removing bacteria and other materials from body tissues and it also transports fat from the small intestines; it eventually empties into the blood, returning tissue fluids into the general body circulation

Diseases Associated with Increased Flow of Fluids Through the Lining of the Intestines (Mucosal Permeability) or Superficial Loss of Tissue of the Lining of the Intestines (Mucosal Ulceration)

- Viral infection/inflammation of the stomach and intestines (known as “gastroenteritis”)—parvovirus and others
- Bacterial infection/inflammation of the stomach and intestines (gastroenteritis)—small intestinal bacterial overgrowth (“SIBO,” condition in which a high number of bacteria are found in the upper small intestine), salmonellosis, and others
- Fungal infection/inflammation of the stomach and intestines (gastroenteritis)—histoplasmosis and others
- Parasitic inflammation of the intestines (known as “enteritis”)—hookworms, whipworms, and others
- Inflammatory bowel disease (“IBD”)
- Adverse food reactions—food allergy, food intolerance
- Mechanical diseases of the intestines (enteropathies)—long-term (chronic) folding of one segment of the intestine into another segment (known as “intussusception”); long-term (chronic) foreign body
- Intestinal cancer—lymphoma, adenocarcinoma
- Superficial loss of tissue on the surface of the lining of the stomach or intestines, frequently with inflammation (known as “ulceration”)

RISK FACTORS

- Disease of the stomach and intestines
- Lymphatic disorders (“lymphatic” refers to vessels within the body that transports lymph, a clear to slightly colored liquid that contains white-blood cells—it serves many functions including removing bacteria from tissues and it also transports fat from the small intestines; it eventually empties into the blood, returning tissue fluids into the general body circulation)
- Heart disease



TREATMENT

HEALTH CARE

- In cases of severely low levels of albumin (a type of protein) in the blood (known as “severe hypoalbuminemia”) and complications due to the hypoalbuminemia, plasma transfusions or use of colloids (intravenous fluids that contain larger molecules that stay within the circulating blood, examples are dextran and hetastarch) should be considered when clinical signs from fluid build-up in tissues (edema or effusion) are severe
- Tapping the abdomen to remove excess fluid (ascites; procedure known as “abdominocentesis”) or tapping the chest to remove excess fluid from the space between the chest wall and lungs (pleural effusion; procedure known as “thoracocentesis”) in cases with problems (such as breathing difficulties) from severe fluid build-up (effusion)

ACTIVITY

- Normal

DIET

- Modified, depending on the underlying cause of protein-losing enteropathy
- A low-fat diet should be used if lymphangiectasia (dilation of the lymphatic vessels in the gastrointestinal tract) is diagnosed or highly suspected
- Elemental diets can be used in patients with severe disease; “elemental diets” are liquid diets that contain amino acids, carbohydrates, low levels of fats, vitamins, and minerals that can be absorbed without the need for digestion

SURGERY

- Low levels of albumin (a type of protein) in the blood (hypoalbuminemia) increases the frequency of postoperative complications, because of slow wound healing
- Some causes of protein-losing enteropathy (such as folding of one segment of the intestine into another segment [intussusception], long-term (chronic) foreign body, and some intestinal cancers) require surgical intervention, even in the face of very low levels of albumin (a type of protein) in the blood (hypoalbuminemia)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a

particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No medications are available to treat protein-losing enteropathy itself
- The underlying cause of protein-losing enteropathy must be treated; medications are selected based on underlying cause
- Medications to remove excess fluid from the body (known as “diuretics,” such as furosemide) have been used by some veterinarians to control fluid build-up under the skin (edema) and between the chest wall and lungs (pleural effusion); however, they may not work well and may be associated with side effects



FOLLOW-UP CARE

PATIENT MONITORING

- Check body weight, serum albumin concentration, and evidence of recurrent clinical signs (such as fluid build-up in the abdomen [ascites], under the skin [edema], and or in the space between the chest wall and lungs [pleural effusion])

POSSIBLE COMPLICATIONS

- Breathing difficulty from fluid build-up in the space between the chest wall and lungs (pleural effusion)
- Severe protein-calorie malnutrition
- Diarrhea that is not responsive to medical treatment

EXPECTED COURSE AND PROGNOSIS

- Prognosis is guarded
- Primary, underlying disease cannot be treated in many cases



KEY POINTS

- “Protein-losing enteropathy” is any disease process that is characterized by excessive loss of proteins from the body into the gastrointestinal tract
- Long-term treatment usually is required; spontaneous cures are rare
- Prognosis is guarded
- Primary, underlying disease cannot be treated in many cases

EOSINOPHILIC GRANULOMA COMPLEX IN CATS AND EOSINOPHILIC GRANULOMAS IN DOGS



BASICS

OVERVIEW

- Cats—"eosinophilic granuloma complex" often is a confusing term for three distinct syndromes: 1) "eosinophilic plaque" (circumscribed, raised, round to oval lesions that frequently are ulcerated; usually located on the abdomen or thighs; lesions contain a type of white blood cell, called an eosinophil); 2) "eosinophilic granuloma" (a mass or nodular lesion containing eosinophils; usually found on the back of the thighs, on the face, or in the mouth); and 3) "indolent ulcer" (circumscribed, ulcerated lesions; most frequently found on upper lip); the three syndromes are grouped together as "eosinophilic granuloma complex" primarily according to their clinical similarities, their frequent simultaneous development, and their positive response to treatment with steroids
- Dogs—"eosinophilic granulomas" are rare; not part of the eosinophilic granuloma complex; specific differences from cats are presented in the following information
- "Eosinophilic" refers to eosinophils, a type of white-blood cell usually involved in allergic responses
- "Granuloma" is a large inflammatory nodule or solid mass
- "Complex" is a group of signs or diseases that have an identifiable characteristic that makes them similar in some fashion

GENETICS

- Unknown
- Several reports of related affected individuals and a study of disease development in a colony of cats indicate that, in at least some individuals, genetic susceptibility (perhaps resulting in an inheritable dysfunction of eosinophils) is a significant component of the disease

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- "Eosinophilic granuloma complex" is restricted to cats
- "Eosinophilic granulomas" occur in dogs and other species, but are not considered part of the eosinophilic granuloma complex

Breed Predilections

- Cats—none
- Eosinophilic granuloma in dogs—Siberian huskies (76% of cases)

Mean Age and Range

- Eosinophilic plaque (circumscribed, raised, round to oval lesions that frequently are ulcerated; usually located on the abdomen or thighs; lesions contain a type of white blood cell, called an eosinophil)—2 to 6 years of age
- Genetically initiated eosinophilic granuloma—less than 2 years of age
- Allergic disorder—over 2 years of age
- Eosinophilic granuloma in dogs—usually less than 3 years of age

Predominant Sex

- Cats—females may be more likely to develop one or more of the syndromes of eosinophilic granuloma complex than are males
- Eosinophilic granuloma in dogs—males (72% of cases)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Distinguishing among the syndromes depends on both clinical signs and microscopic findings
- Lesions of more than one syndrome may occur simultaneously; lesions of all three syndromes may develop spontaneously and suddenly (acutely)
- Development of eosinophilic plaques (circumscribed, raised, round to oval lesions that frequently are ulcerated; usually located on the abdomen or thighs; lesions contain a type of white blood cell, called an eosinophil) can be preceded by periods of sluggishness (lethargy)
- A seasonal incidence is possible
- Signs vary in intensity—they may increase and decrease over time (known as a "waxing and waning" course), which is common in all three syndromes
- Eosinophilic plaques—loss of hair (known as "alopecia"), reddened skin (known as "erythema"), patches of loss of superficial layers of skin (known as "erosive patches") or well-demarcated, steep-walled thickened, raised, flat-topped areas that are slightly higher than normal skin (known as "plaques"); usually occur in the inguinal or perineal (area between the anus and external genitalia) areas, along the

thighs, lower abdomen, and under the front legs, near the chest; frequently moist or glistening; may have enlarged lymph nodes near the area of the eosinophilic plaques

- Eosinophilic granulomas (masses or nodular lesions containing eosinophils)—occur in a distinctly linear orientation (“linear granuloma”) on the back part of the thigh, or as individual lesions or multiple lesions that are coming together, located anywhere on the body; ulcerated with a “cobblestone” or coarse pattern; white or yellow, possibly representing collagen degeneration; lip margin and chin swelling (“pouting”); footpad swelling, pain, and lameness (most common in cats under 2 years of age); ulcers of the mouth (especially on the tongue, palate, and palatine arches)—cats with ulcers of the mouth may have difficulty swallowing (known as “dysphagia”), have bad breath (known as “halitosis”), and may drool
- Lesion development may stop spontaneously in some cats, especially with the inheritable form of eosinophilic plaque
- Indolent ulcers—classically concave and firm or hardened ulcerations with a granular, orange-yellow color, confined to the upper lips
- Eosinophilic granuloma in dogs—ulcerated, thickened, raised, flat-topped areas that are slightly higher than normal skin (plaques) and masses; dark or orange color

CAUSES

- Allergy—flea or insect (such as mosquito-bite) allergy, food allergy, and atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems)
- Inherited dysfunction of eosinophils is a possible cause
- Eosinophilic granuloma in dogs—unknown cause; increased sensitivity or reaction in the skin to the presence of a foreign material (known as “hypersensitivity”) often is suspected (such as to an insect bite)



TREATMENT

HEALTH CARE

- Most patients can be treated as outpatients, unless severe disease of the mouth prevents adequate fluid intake
- Try to identify and eliminate offending allergen(s) before providing medical intervention; “allergens” are substances to which the animal has developed an allergy
- “Allergy shots” (known as “hyposensitization”) in cats that have tested positive on skin tests for allergies—may be successful in 60% to 73% of cases; preferable to long-term steroid administration
- Avoid excessive grooming, which may damage the skin lesions

ACTIVITY

- No restrictions

DIET

- No restrictions, unless a food allergy is suspected

SURGERY

- Skin biopsy to obtain samples for microscopic evaluation of lesions
- Surgical removal of a lesion may be performed in some cases



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

EOSINOPHILIC GRANULOMA COMPLEX IN CATS

Eosinophilic Plaque (circumscribed, raised, round to oval lesions that frequently are ulcerated)

- Injectable methylprednisolone—most common treatment
- Steroids—ongoing treatment with prednisolone required to control lesions; other drugs: dexamethasone and triamcinolone

Eosinophilic Granuloma (a mass or nodular lesion containing eosinophils)

- Steroids administered by injection or by mouth—most common treatment
- Combination of steroids and medications to decrease the immune response (known as “immunosuppressive drugs”)—for severe lesions; example of an immunosuppressive drug, chlorambucil

- Antibiotics—may be beneficial, if lesions are infected

Indolent Ulcer (circumscribed, ulcerated lesions; most frequently found on upper lip)

- Steroids administered by injection or by mouth
- α -Interferon—administered daily in cycles of 7 days on, 7 days off; limited success; side effects rare; no specific treatment monitoring required
- Antibiotics—clindamycin, cephalexin, or amoxicillin-clavulanate; effective in some cases; preferable to long-term steroid administration; response may be the result of the anti-inflammatory activity of these drugs rather than their primary antibiotic properties

Other Therapies

- Radiation and modification of the immune response (known as “immunomodulation”), such as with levamisole or bacterin injections—occasional reports of success
- Carbon dioxide (CO₂) laser—may offer relief from individual or painful lesions, especially those in the mouth
- Application of steroid ointments onto the lesions directly (known as “topical treatment”) may help with isolated lesions, but rarely is practical
- Doxycycline, an antibiotic
- Cyclosporine (medication to decrease the immune response) has been used; mixed results, but encouraging success reported
- Megestrol acetate—can be effective in rare cases; not recommended because of the severity of possible side effects

EOSINOPHILIC GRANULOMA IN DOGS

- Steroids administered by mouth—prednisone



FOLLOW-UP CARE

PATIENT MONITORING

- Pets receiving steroids—baseline and follow-up blood work (complete blood counts [CBCs] and serum chemistry profiles) and urinalyses with bacterial culture and sensitivity testing of urine
- Pets receiving medications to decrease the immune response (immunosuppressive drugs)—frequent complete blood counts ([CBCs], biweekly at first, then monthly or bimonthly as therapy continues) to monitor for bone-marrow suppression leading to low red-blood cell and low white-blood cell counts; routine serum chemistry profiles and urinalyses with bacterial culture and sensitivity testing of urine (monthly at first, then every 3 months) to monitor for complications (such as kidney disease, diabetes mellitus, and urinary tract infection)

EXPECTED COURSE AND PROGNOSIS

- If a primary cause (allergy) can be determined and controlled, lesions should resolve permanently, unless the animal re-encounters the offending allergen (substances to which the animal has developed an allergy)
- Most lesions increase and decrease over time (wax and wane), with or without therapy; thus an unpredictable schedule of recurrence should be anticipated
- Drug dosages should be tapered to the lowest possible level (or discontinued, if possible), once the lesions have resolved; changes in drug dosage should be at the direction of your pet’s veterinarian
- Lesions in cats with the inheritable disease may resolve after several years



KEY POINTS

- Possible allergic or inherited causes
- Most lesions increase and decrease over time (wax and wane), with or without therapy; thus an unpredictable schedule of recurrence should be anticipated
- In some cases, a decision may be made to postpone medical intervention, unless severe lesions develop

IDIOPATHIC EPILEPSY (SEIZURE DISORDER OF UNKNOWN CAUSE)



BASICS

OVERVIEW

- “Idiopathic” is the medical term for a disease or disorder of unknown cause; “epilepsy” is a brain disorder, in which the animal has sudden, recurring attacks, with or without loss of consciousness
- “Idiopathic epilepsy” is a brain disorder characterized by recurrent seizures in the absence of structural brain lesion; it is age-related and assumed to have a genetic basis

GENETICS

- Dogs—genetic in many breeds; familial (runs in certain families or lines of animals) susceptibility reported for the beagle, keeshond, Belgian Tervuren, golden retriever, Labrador retriever, vizsla and Shetland sheepdog.
- Polygenic recessive mode of inheritance suggested in the Bernese mountain dog and Labrador retriever; autosomal recessive trait proposed in the vizsla and Irish wolfhound; partially penetrant autosomal recessive trait in the English springer spaniel; genetic cause suspected in the Finnish spitz

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and rarely cats

Breed Predilections

- Beagles, all shepherds (German shepherd dog, Australian shepherd, Belgian Tervuren), boxers, cocker spaniels, collies, border collies, dachshunds, golden retrievers, Irish setters, Irish wolfhound, keeshonden, Labrador retrievers, poodles (all sizes), St. Bernards, Shetland sheepdogs, Siberian huskies, English springer spaniels, Welsh corgis; wire fox terriers

Mean Age and Range

- Range—6 months to 5 years of age
- Prevalence—10 months to 3 years of age

Predominant Sex

- Males are more likely to have idiopathic epilepsy than females in the Bernese mountain dog

SIGNS/OBSERVED CHANGES in the ANIMAL

- Seizures may be generalized from onset, or have a short aura (focal onset) with rapid generalization of seizure activity; an “aura” is a sensation that precedes a seizure—for example, the animal appears frightened, dazed, seeks attention, or hides
- Presence of an aura is frequent, preceding the generalized seizure activity
- Seizures are mainly focal (that is, involve localized areas of the brain) in the Finnish spitz
- Seizures—most occur while the patient is resting or asleep; often at night or in early morning; frequency tends to increase if left untreated; affected animal falls on its side, becomes stiff, chomps its jaw, salivates profusely, urinates, defecates, vocalizes, and paddles with all four limbs in varying combinations; seizure activity is of short duration (30 to 90 seconds)
- Behavior following the seizure (known as “post-ictal behavior”)—periods of confusion and disorientation; aimless wandering, compulsive behavior, blindness, pacing; frequent increased thirst (known as “polydipsia”) and increased appetite (known as “polyphagia”); recovery immediate or may take up to 24 hours following the seizure
- Dogs with established epilepsy may have cluster seizures at regular intervals of 1 to 4 weeks; particularly evident in large-breed dogs
- No asymmetry in movements should be observed during seizure, such as twitching more pronounced on one side, limb contractions on one side, circling just before or after the seizure
- Physical examination findings generally are normal; patients usually have recovered by time of presentation to the veterinarian

CAUSES

- Unknown cause (idiopathic)
- Genetic in some breeds

RISK FACTORS

- Known epilepsy in the parents



TREATMENT

HEALTH CARE

- Initiate treatment at the second generalized seizure, or if sudden (acute) cluster seizures or sudden (acute) repeated or prolonged seizure activity (known as “status epilepticus”) occur
- Inpatient—seizure disorder requires constant monitoring; cluster seizures (more than 1 seizure/24 hours) repeated or prolonged seizure activity (status epilepticus); treat early and aggressively
- Outpatient—recurrence of isolated seizures
- Other treatments: acupuncture, vagal nerve stimulation, transcranial magnetic motor stimulation

ACTIVITY

- Avoid swimming, to prevent drowning

DIET

- Most dogs on long-term (chronic) medications to control seizures (known as “antiepileptic drugs” or “anticonvulsants”) become overweight; monitor weight closely; add a weight-reducing program as necessary
- Potassium bromide (KBr) treatment to control seizures—patients should have steady levels of salt in their diets to maintain therapeutic levels of KBr in the serum; an increase in salt causes an increase in bromide excretion preferentially over chloride, with subsequent decreased serum KBr levels; alternatively, a decreased salt content leads to increased KBr serum levels
- If the epileptic dog treated with potassium bromide (KBr) requires a diet change, take into consideration salt content difference
- Avoid salty treats in dogs treated with potassium bromide (KBr)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Phenobarbital and Potassium Bromide (KBr)

- Phenobarbital requires 12 to 15 days of treatment to reach steady levels in the serum; potassium bromide (KBr) requires 3 to 4 months of treatment to reach steady levels in the serum and levels vary with salt concentration in diet; in dogs predisposed to epilepsy that is not responsive to medical treatment, both drugs may be initiated at once, in which case, the combine treatment may produce a beneficial and synergistic effect
- Phenobarbital—traditional first-line drug; evaluate serum levels 12 to 15 days after onset of treatment and after changes in dosage; evaluate levels again 4 weeks after initiation of treatment; increase dosage if needed and recheck levels every two weeks until optimal therapeutic range is reached
- Potassium bromide (KBr)—alternative first-line drug; may be added to treatment for patients started on phenobarbital that continue to have seizures at a frequency of more than one every 8 to 12 weeks

Diazepam

- To treat ongoing seizures; dogs with cluster seizures or repeated or prolonged seizure activity (status epilepticus)
- Inpatient treatment—administer diazepam intravenously
- Reinstate medications administered by mouth (oral medication) as soon as possible; the oral dosage of phenobarbital and/or potassium bromide (KBr) may be increased as directed by your pet’s veterinarian, if the serum drug levels before emergency treatment were inadequate
- Outpatient treatment—for dogs known to have cluster seizures, as directed by your pet’s veterinarian

Other Drugs

- With the use of multiple drugs (known as “polypharmacy”) to control seizures, initiate add-on medications gradually to avoid sedation
- Levetiracetam or gabapentin; both drugs are well tolerated, safe to add to phenobarbital and potassium bromide (KBr) and have generic formulation available
- Zonisamide; well tolerated; safe to add to phenobarbital and potassium bromide (KBr); high cost; not available in Canada
- Felbamate; not readily available in Canada
- Clorazepate; clonazepam



FOLLOW-UP CARE

PATIENT MONITORING

- Serum drug levels—essential to monitor therapeutic levels of drugs in the blood
- Phenobarbital—measure 2 and 4 weeks after initiating therapy; adjust oral dose as needed; repeat until the optimal serum levels are reached; with long-term (chronic) administration of phenobarbital, monitor blood work (complete blood count [CBC], serum chemistry profile, and drug levels) every 6 to 12 months to evaluate potential side effects and serum levels of the drugs
- Potassium bromide (KBr)—elimination rate depends on concentration of salt in the food; measure serum levels (along with phenobarbital levels) 4 to 6 weeks after initiating treatment
- Carefully monitor older dogs with kidney insufficiency that are on potassium bromide (KBr) treatment
- If the epileptic dog treated with potassium bromide (KBr) requires a diet change, take into consideration salt content difference and monitor accordingly

PREVENTIONS AND AVOIDANCE

- Abrupt discontinuation of medication(s) to control seizures may precipitate seizures
- Avoid salty treats in dogs treated with potassium bromide (KBr)

POSSIBLE COMPLICATIONS

- Phenobarbital-induced high serum alkaline phosphatase (“alkaline phosphatase” is a normal enzyme found in several types of cells, including liver cells; increased levels of alkaline phosphatase may indicate abnormal liver function)—occurs frequently; may be an early sign of liver toxicity, but is of less concern if another liver enzyme (alanine aminotransferase [ALT]) on the blood work is within the normal range
- Phenobarbital-induced liver toxicity—occurs after long-term (chronic) treatment with serum phenobarbital levels in the middle to upper therapeutic range; may be subtle in onset; the only biochemical abnormality may be a decrease in albumin (a protein in the blood, that is produced by the liver)
- Higher incidence of inflammation of the pancreas (known as “pancreatitis”) in patients treated with phenobarbital and/or potassium bromide (KBr); once pancreatitis develops, recurrence is frequent
- Phenobarbital: rare decreased production of blood cells by the bone marrow (known as “bone-marrow suppression”) with severe low neutrophil count (neutrophils are a specific type of white-blood cell that fight infection; low neutrophil count is known as “neutropenia”) and generalized bacterial infection (known as “sepsis”) may develop early in the course of treatment; if occurs, discontinue drug as directed by your pet’s veterinarian
- Unexpected hyperexcitability may result with phenobarbital treatment; if occurs, discontinue drug as directed by your pet’s veterinarian
- Potassium bromide (KBr)—patient may be unsteady while managing stairs

EXPECTED COURSE AND PROGNOSIS

- Treatment for life
- Some dogs are well controlled with the same drug and dosage for years; others remain poorly controlled despite the use of multiple medications (polypharmacy)
- Medication(s) to control seizures (antiepileptic treatment)—decreases frequency, severity, and length of seizures; perfect control rarely achieved
- In many young, large-breed dogs, seizures may continue despite adequate treatment
- Lack of response to treatment may develop
- Patient may develop repeated or prolonged seizure activity (status epilepticus) and die



KEY POINTS

- Animals usually do not die after a seizure; however, severe cluster seizures and repeated or prolonged seizure activity (status epilepticus) are life-threatening emergencies requiring immediate veterinary medical attention
- Prevent the patient from injuring itself on surrounding objects during a seizure
- Keep a calendar of the seizures noting date, time, length and severity of the seizures, as an objective way to assess response to

treatment

- Once treatment is instituted, the patient will require medication for life in most cases

OVERFLOW OF TEARS (EPIPHORA)



BASICS

OVERVIEW

- Abnormal overflow of tears

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Depend on cause; see **CAUSES**

Mean Age and Range

- Depend on cause; see **CAUSES**

SIGNS/OBSERVED CHANGES in the ANIMAL

- Overflow of tears
- Tear drainage and/or staining on face
- Other signs depend on cause

CAUSES

Overproduction of Tears Secondary to Eye Irritants

Congenital (present at birth)

- Two rows of eyelashes present in a single eyelid (known as “distichiasis”) or abnormal eyelashes that turn inward, against the cornea (the clear part of the eye, located in the front of the eyeball; condition known as “trichiasis”)—young shelties; shih tzus; Lhasa apsos; cocker spaniels; miniature poodles
- Eyelid is curled inward toward the eyeball (known as “entropion”)—Chinese shar peis; chow chows
- Absence of the eyelid (known as “eyelid agenesis”)—domestic shorthairs (cats)

Acquired (develop later in life, after birth)

- Corneal or conjunctival foreign bodies—usually young, large-breed, active dogs; “corneal” refers to the “cornea,” the clear outer layer of the front of the eye; “conjunctival” refers to the “conjunctiva,” the moist tissues of the eye
- Eyelid tumors—old dogs (all breeds)
- Inflammation of the eyelids (known as “blepharitis”)—infectious or immune-mediated
- Inflammation of the moist tissues of the eye (known as “conjunctivitis”)—infectious or immune-mediated
- Disorder of the cornea (the clear outer layer of the front of the eye) characterized by the presence of ulcers, with or without inflammation (condition known as “ulcerative keratitis”)
- Inflammation of the front part of the eye, including the iris (known as “anterior uveitis”); the “iris” is the colored or pigmented part of the eye
- Disease of the eye, in which the pressure within the eye is increased (known as “glaucoma”)

Eyelid Abnormalities or Poor Eyelid Function

- Tears never reach the opening to the outflow portion of the drainage system (known as the “nasolacrimal puncta”) that normally moves tears to the nasal passages, but instead spill over the eyelid margin

Congenital (present at birth)

- Too large an opening of the eyelids, causing increased exposure of the eyeball (known as “macropalpebral fissures”)—in short-nosed, flat-faced (brachycephalic) breeds
- Eyelid is turned outward, away from the eyeball (known as “ectropion”)—Great Danes; bloodhounds; spaniels
- Eyelid is curled inward, toward the eyeball (entropion)—especially of the lower lid, closest to the nose

Acquired (develop later in life, after birth)

- Post-traumatic eyelid scarring
- Facial nerve paralysis

Blockage of the Nasolacrimal Drainage System (the drainage system that normally moves tears to the nasal passages)

Congenital (present at birth)

- Lack of normal openings on the eyelids into the tear drainage system (known as “imperforate puncta”)—cocker spaniels, bulldogs, poodles
- Extra openings into the tear drainage system, located in abnormal positions (known as “ectopic nasolacrimal openings”)—openings along the side of the face below the corner of the eye, closest to the nose
- Lack of openings from the tear drainage system into the nose (known as “nasolacrimal atresia”)

Acquired (develop later in life, after birth)

- Inflammation of the nose (known as “rhinitis”) or inflammation of the sinuses (known as “sinusitis”)—causes swelling adjacent to the tear drainage system (nasolacrimal duct)
- Trauma or fractures of bones in the face (lacrimal or maxillary bones)
- Foreign bodies—grass awns; seeds; sand; parasites
- Tumors of the third eyelid, the moist tissues of the eye (conjunctiva), eyelids, nasal cavity, maxillary bone in the face, or sinuses located around the eyes
- Inflammation of the nasolacrimal sac, part of the tear drainage system (condition known as “dacryocystitis”)

RISK FACTORS

- Breeds prone to congenital (present at birth) eyelid abnormalities (see **CAUSES**)
- Active, outdoor dogs—at risk for foreign bodies



TREATMENT

HEALTH CARE

- Remove cause of eye irritation—remove foreign body from the moist tissues of the eye (conjunctiva) or the clear outer layer of the front of the eye (cornea); treatment of the primary eye disease (such as inflammation of the moist tissues of the eye [conjunctivitis]; disorder of the cornea characterized by the presence of ulcers, with or without inflammation [ulcerative keratitis]; and inflammation of the iris and other areas in the front part of the eye [known as “uveitis”]); freezing procedure (known as “cryosurgery”) or removal of hair by electrolysis (procedure known as “electroepilation”) to treat the condition in which two rows of eyelashes are present in a single eyelid (distichiasis) or surgical repair of abnormal eyelids
- Treat primary lesion (such as a third eyelid mass, mass in the nose or sinus, and infection) that is blocking drainage of tears—successful management may allow normal tear flow through the tear drainage system (nasolacrimal flow) to resume
- Patients with inflammation of the nasolacrimal sac, part of the tear drainage system (dacryocystitis), may need a catheter placed in the nasolacrimal duct to hold the duct open and to prevent scar formation

SURGERY

Lack of Normal Openings on the Eyelids into the Tear Drainage System (Imperforate Puncta)

- Surgical opening of the opening (puncta) into the tear drainage system
- Opening (puncta) closed by scar tissue between the eyelid and the eyeball (known as “symblepharon”) caused by severe inflammation of the moist tissues of the eye (conjunctivitis), such as caused by feline herpesvirus in cats—surgically open the puncta
- Recurrent disease—may be necessary to suture Silastic® tubing in place to prevent scar formation

Blocked Openings of the Tear Drainage System into the Nose

- Surgical procedure to create an opening to drain the tears into the nasal cavity



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Topical (applied directly to the eye) broad-spectrum antibiotic eye solutions—while waiting for results of diagnostic tests (such as bacterial culture and sensitivity testing; diagnostic X-rays); may try neomycin, gramicidin, polymyxin B triple antibiotic solutions, or chloramphenicol solution
- Inflammation of the nasolacrimal sac, part of the tear drainage system (dacryocystitis)—topical antibiotics based on bacterial culture and sensitivity test results; continued for at least 21 days

- Tetracycline is an antibiotic, given by mouth—may help reduce tear staining of facial hair below the eye, for example as seen in white poodles; staining recurs when tetracycline treatment is discontinued



FOLLOW-UP CARE

PATIENT MONITORING

Inflammation of the Nasolacrimal Sac, Part of the Tear Drainage System (Dacryocystitis)

- Reevaluate every 7 days until the condition is resolved
- Continue treatment for at least 7 days after resolution of clinical signs to help prevent recurrence
- Problem persists more than 7 to 10 days with treatment or recurs soon after cessation of treatment—indicates a foreign body or persistent infection may be involved; requires further diagnostics

Surgical Procedure to Create an Opening to Drain Tears into the Nasal Cavity

- Tubing—reevaluate every 7 days to ensure it remains intact; may need to resuture if it becomes loosened or dislodged
- After tubing has been removed—reevaluate in 14 days; fluorescein dye will be placed on the eye and the flow of tears through the tear drainage system will be checked by examining the nose for appearance of the fluorescein dye (indicating that tears are flowing through the drainage system)
- X-ray contrast study of the tear drainage system—repeated 3 to 4 months after surgery to evaluate size of the nasal opening; repeated for recurrence or with no appearance of fluorescein dye through the tear drainage system

POSSIBLE COMPLICATIONS

- Recurrence—most common complication; caused by recurrence of cause of eye irritation, recurrence of inflammation of the nasolacrimal sac, part of the tear drainage system (dacryocystitis), or closure of the surgical openings created to allow tears to drain into the nasal cavity

EXPECTED COURSE AND PROGNOSIS

- Depend on cause
- The patient treated for a primary lesion (such as a third eyelid mass, mass in the nose or sinus, and infection) that had been blocking drainage of tears is susceptible to blockage of the tear drainage system (nasolacrimal obstruction) and recurrence is common; early detection and intervention provide a better long-term prognosis



KEY POINTS

- Abnormal overflow of tears
- Remove cause of eye irritation
- Treat primary lesion (such as a third eyelid mass, mass in the nose or sinus, and infection) that is blocking drainage of tears—successful management may allow normal tear flow through the tear drainage system (nasolacrimal flow) to resume
- Recurrence of abnormal overflow of tears is the most common complication

BLEEDING FROM THE NOSE (EPISTAXIS)



BASICS

OVERVIEW

- Bleeding from the nose

SIGNALMENT/DESCRIPTION of ANIMAL

- Depends on underlying cause

SIGNS/OBSERVED CHANGES in the ANIMAL

- Bleeding from one or both nostrils (nasal hemorrhage)
- Sneezing
- May see bleeding from other areas of the body if nose bleed related to blood-clotting disorder (known as “coagulopathy”), such as blood in the stool (hematochezia); dark black, tarry stool due to the presence of digested blood (melena); blood in the urine (hematuria)
- May see bruising or blood-filled swellings (hematomas) if nose bleed related to blood-clotting disorder (coagulopathy)
- May have vision disorders due to bleeding in the retina (retinal hemorrhages) with blood-clotting disorders (coagulopathy) or high blood pressure (hypertension)

CAUSES

Bleeding from the nose results from one of three abnormalities—bleeding disorders or blood-clotting disorders (coagulopathies); presence of a mass or space-occupying lesion; blood vessel (vascular) or generalized (systemic) disease

Bleeding Disorders or Blood-Clotting Disorders (Coagulopathies)

Low Number of Platelets or Thrombocytes in the Blood (known as “Thrombocytopenia”)

- Immune-mediated disease—thrombocytopenia for unknown reason (so called, “idiopathic disease”); systemic lupus erythematosus (SLE); drug reaction; modified-live vaccine (MLV) reaction
- Infectious disease—*Ehrlichia* infection; Rocky Mountain spotted fever; *Babesia* infection; feline leukemia virus (FeLV)- or feline immunodeficiency virus (FIV)-related illness
- Bone-marrow disease—cancer; aplastic anemia, where the bone marrow is not generating blood cells; infectious disease (fungal, rickettsial, or viral)
- Disorders that accompany cancer (known as “paraneoplastic disorders”)
- Blood clotting disorder—disseminated intravascular coagulopathy (DIC), a bleeding problem in which clotting factors are activated and clotting factors and platelets are used up

Abnormal Function of Platelets or Thrombocytes in the Blood (known as “Thrombopathia”)

- Congenital (present at birth)—von Willebrand’s disease (bleeding disorder caused by lower than normal levels of factor VII—one of the ingredients required to clot blood); abnormally functioning platelets (thrombopathia)
- Acquired (present after birth)—nonsteroidal anti-inflammatory drugs (NSAIDs); increased levels of globulin, a body protein, in the blood (known as “hyperglobulinemia,” may be seen with *Ehrlichia* infection or multiple myeloma); increased concentration of nitrogenous waste products including urea in the blood (known as “uremia”); blood clotting disorder—disseminated intravascular coagulopathy (DIC)

Blood-Clotting (Coagulation) Factor Defects

Blood-clotting (coagulation) factors are present in the plasma of the blood. They are ingredients that come together in a certain order to produce a clot. The clotting factors are identified by Roman numerals, I through XII. If one or more blood-clotting factor is present in too low a level or if the blood clotting factors have been used up, clotting will not occur normally and bleeding will result. The amount of bleeding varies.

- Congenital (present at birth): hemophilia A (factor VIIIc deficiency) and hemophilia B (factor IX deficiency)
- Acquired (present after birth): poisoning with agents used to kill rodents (mice, rats)—these agents (known as “anticoagulant rodenticides,” such as warfarin) prevent blood clotting; liver disease; and disseminated intravascular coagulopathy (DIC), a bleeding problem in which clotting factors are activated and clotting factors and platelets are used up

Mass or Space-Occupying Lesion

- Foreign body
- Trauma
- Infection—fungal (*Aspergillus*, *Cryptococcus*, and *Rhinosporidium*); viral or bacterial; usually see blood-tinged nasal discharge rather than obvious bleeding
- Cancer

- Dental disease—abnormal opening between the mouth and nose (known as an “oronasal fistula”), tooth-root abscess

Blood Vessel (Vascular) or Generalized (Systemic) Disease

- High blood pressure (known as “hypertension”)—kidney disease; excessive production of thyroid hormone (known as “hyperthyroidism”); excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); high blood pressure caused by unknown reason (known as “idiopathic disease”)
- Increase in proteins in the serum of the blood (known as “hyperviscosity”)—multiple myeloma (cancer of the bone marrow in which abnormal proteins are produced); *Ehrlichia* infection; increased number of red blood cells (known as “polycythemia”)
- Inflammation of the blood vessels (known as “vasculitis”)—immune-mediated and rickettsial diseases

RISK FACTORS

Coagulopathy

- Immune-mediated disease—young to middle-aged, small to medium female dogs
- Infectious disease—dogs living in or traveling to areas where other dog or animals are carrying the infection-causing organism; tick exposure
- Abnormality of the platelets (thrombasthenia)—otter hounds
- Abnormal functioning of the platelets (thrombopathia)—basset hounds, spitz
- von Willebrand’s disease—Doberman pinschers, Airedale terriers, German shepherd dogs, Scottish terriers, Chesapeake Bay retrievers, and many other breeds; cats
- Hemophilia A—German shepherd dogs and many other breeds; cats
- Hemophilia B—Cairn terriers, coonhounds, St. Bernards, and other breeds; cats

Mass or Space-Occupying Lesion

- Aspergillosis—German shepherd dog, Rottweiler
- Cancer—long-nosed dogs (known as “dolicocephalic breeds”)



TREATMENT

HEALTH CARE

- Blood-clotting disorder (coagulopathy)—usually inpatient
- Mass or space-occupying lesion or blood vessel (vascular) or generalized (systemic) disease—outpatient or inpatient, depending on the disease and its severity
- Recognize signs of serious bleeding (such as weakness, collapse, pallor, and obvious blood loss)
- Whole-blood or packed red blood-cell (RBC) transfusion—may be needed with severe anemia

Coagulopathy

- von Willebrand’s disease—plasma or cryoprecipitate (a product of blood plasma that has been cooled to increase levels of clotting factors) for acute bleeding
- Hemophilia A—plasma or cryoprecipitate for acute bleeding; no long-term treatment
- Hemophilia B—plasma for acute bleeding; no long-term treatment
- Rat poisoning (anticoagulant rodenticide poisoning)—plasma for acute bleeding; vitamin K
- Liver disease and disseminated intravascular coagulopathy (DIC)—treat and support the underlying cause; plasma may be beneficial
- Discontinue all nonsteroidal anti-inflammatory drugs (NSAIDs)
- Increased levels of globulin in the blood (hyperglobulinemia)—plasmapheresis (medical process in which whole blood is removed from the body, the blood cells are separated from the fluid portion of the blood and then are put into a sterile fluid and transfused back into the body)
- Increased number of red blood cells (known as “polycythemia”)—phlebotomy (medical procedure in which whole blood is removed from the body)

Mass or Space-Occupying Lesion

- Radiation therapy—nasal tumors; various response rates depending on tumor type
- Removal of foreign body

ACTIVITY

- Minimize activity or stimuli that may lead to or increase bleeding episodes

DIET

- Depends on underlying cause
- Weight reduction and sodium restriction may be indicated in treatment of high-blood pressure (hypertension)

SURGERY

- Removal of foreign body
- Fungal infection of the nose (known as “fungal rhinitis,” such as caused by *Aspergillus* and *Rhinosporidium*) may require surgical removal of part of the space-occupying lesion (known as “debulking”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Coagulopathy

- Immune-mediated disease—prednisone; additional drugs may be used for cases that do not respond to prednisone alone, such as azathioprine, cyclosporine, danazole
- Infectious disease—Rickettsial disease: doxycycline; *Babesia* infection: imidocarb
- Bone-marrow cancer—chemotherapy
- Abnormal platelet function or platelets (thrombopathia or thrombasthenia)—no treatment in most cases
- Desmopressin acetate or DDAVP is a synthetic antidiuretic hormone; DDAVP may help control bleeding owing to von Willebrand’s disease
- Rat poisoning (anticoagulant rodenticide poisoning)—plasma for acute bleeding; vitamin K, length of time for vitamin K treatment based on type of rat poison to which animal was exposed

Mass or Space-Occupying Lesion

- Serious bleeding—control with cage rest and acepromazine to lower blood pressure and promote clotting if the patient does not have a decrease in blood volume (known as “hypovolemia”); instillation of Neosynephrine® or dilute epinephrine into the nose may help (these drugs promote narrowing of the blood vessels, known as “vasoconstriction”)
- Bacterial infection—antibiotics; based on culture and sensitivity testing
- Fungal infection—topical treatment with clotrimazole or enilconazole; dapsone following surgery for rhinosporidiosis

Blood Vessel (Vascular) or Generalized (Systemic) Disease

- Increase in proteins in the serum of the blood (known as “hyperviscosity”)—treat underlying disease (such as *Ehrlichia* infection and multiple myeloma)
- Inflammation of the blood vessels (vasculitis)—prednisone for immune-mediated disease; doxycycline for rickettsial disease

High-Blood Pressure (Hypertension)

- Treat underlying disease—kidney disease, excessive production of thyroid hormone (known as “hyperthyroidism”); excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Reduce weight
- Restrict sodium
- Calcium channel blockers—amlodipine; diltiazem
- Angiotensin-converting enzyme (ACE) inhibitors—benazepril; enalapril
- β -blockers—propranolol; atenolol
- Diuretics—hydrochlorothiazide; furosemide



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on underlying cause; discuss with your pet’s veterinarian
- Platelet count for patients with low platelet counts (thrombocytopenia)
- Blood-clotting (coagulation profile) for patients with blood-clotting (coagulation) factor defects
- Blood pressure for patients with high blood pressure (hypertension)
- Clinical signs

PREVENTIONS AND AVOIDANCE

- Depends on underlying cause; discuss with your pet's veterinarian
- Prevent exposure to rat poisons (anticoagulant rodenticide poisons)

POSSIBLE COMPLICATIONS

- Anemia and collapse (rare)

EXPECTED COURSE AND PROGNOSIS

- Depends on underlying cause



KEY POINTS

- Bleeding from the nose results from one of three abnormalities—bleeding disorders or blood-clotting disorders (coagulopathies); presence of a mass or space-occupying lesion; blood vessel (vascular) or generalized (systemic) disease
- May see bleeding from other areas of the body if nose bleed related to blood-clotting disorder (known as “coagulopathy”)

NARROWING OF THE ESOPHAGUS (ESOPHAGEAL STRICTURE)



BASICS

OVERVIEW

- The esophagus is the tubular organ that runs from the throat to the stomach; an esophageal stricture is an abnormal narrowing of esophageal lumen (the inner open space of the esophagus)

GENETICS

- No apparent genetic basis

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Any age; strictures secondary to cancer tend to occur in middle-aged to older animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs are related to the severity and extent of narrowing or stricture
- Regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth)
- Liquid meals often tolerated better than solid meals
- Difficulty swallowing (known as “dysphagia”)—seen with upper esophageal strictures
- Drooling or salivation
- Howling, crying, or yelping during swallowing when the animal has active inflammation of the esophagus (known as “esophagitis”)
- Good appetite initially; eventually, lack of appetite (anorexia) with progressive esophageal narrowing and inflammation
- Weight loss and malnutrition as the disease progresses
- May see aspiration pneumonia with progressive regurgitation and difficulty swallowing (dysphagia)
- Weight loss to severe weight loss with muscle wasting (known as “cachexia”)—in animals with chronic or advanced stricture
- Excessive production of saliva and drooling and/or pain on feeling (palpation) the neck and esophagus—may be seen in animals with inflammation of the esophagus (esophagitis) at the same time as the stricture is present
- Abnormal lung or breathing sounds (such as wheezes and coughing)—may be detected in animals with aspiration pneumonia

CAUSES

- Backward or reverse flow of stomach contents into the esophagus (known as “gastroesophageal reflux”) during anesthesia—most common
- Ingestion of chemical irritants
- Persistent vomiting
- Esophageal retention of pills and capsules
- Backward or reverse flow of stomach contents into the esophagus, unrelated to anesthesia (known as “gastroesophageal reflux disease”)
- Esophageal foreign body
- Esophageal surgery
- Cancer
- Mass lesion (known as a “granuloma”) secondary to a parasite, *Spirocerca lupi*; occasionally seen in the southeastern United States

RISK FACTORS

- Poor preparation (either not fasted adequately or prolonged fasting) prior to anesthesia places some patients at risk for backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux), leading to inflammation of the esophagus (esophagitis), and subsequently to scarring or narrowing of the esophagus (stricture formation)
- Certain drugs used prior to anesthesia (such as diazepam, atropine, pentobarbital, phenothiazine-derivative tranquilizers)—decrease the pressure of the muscle that closes the opening between the esophagus and stomach (known as the “gastroesophageal sphincter”) and can

result in the backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux)



TREATMENT

HEALTH CARE

- Inpatient management initially
- May discharge patients from the hospital after addressing hydration needs, achieving dilation of the affected segment, and initiating any needed treatment for aspiration pneumonia and inflammation of the esophagus (esophagitis)
- Intravenous fluids—may be needed to correct hydration status
- Medications—give by injection following dilation procedures, to facilitate healing
- Oxygen—may be needed for patients with severe aspiration pneumonia

ACTIVITY

- Unrestricted for most cases
- May be limited for patients with aspiration pneumonia

DIET

- Withhold feeding patients by mouth that have severe inflammation of the esophagus (esophagitis) and following dilation procedures
- Temporary feeding tube may be placed at the time of esophageal dilation as a means of providing continual nutritional support
- Give liquid meals when restarting feeding by mouth

SURGERY

- Dilate the narrowed opening of the esophagus by inserting one or more cylindrical medical instruments to gradually open up the narrowed area (known as “Bougienage tube dilation”)
- Mechanical dilation using balloon catheter to open up the narrowed area, with observation of the procedure and esophagus using a special lighted medical instrument called an “endoscope” (general term for procedure is “endoscopy”) or using special X-ray equipment called a “fluoroscope” that allows one to see movement of the balloon (procedure is “fluoroscopy”); perform endoscopy after dilation to assess damage to the lining of the esophagus; redilation at 1- to 2-week intervals may be necessary until stricture is resolved
- Surgical removal of the narrowed section of the esophagus—reportedly has less than a 50% success rate and often is associated with substantial postoperative complications
- Other surgical methods



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Administer medications by injection following dilation procedures and if severe inflammation of the esophagus (esophagitis) is present
- When administering medications by mouth is resumed, dissolve medications in water and give by syringe or give directly via a feeding tube to ensure that they reach the stomach
- Anti-inflammatory dosage of corticosteroids (such as prednisone)—may help prevent scarring and restricture during the healing phase
- Injections of triamcinolone directly into the area where the stricture was located, after dilation of esophageal stricture, may be helpful in decreasing esophageal scarring
- Sucralfate suspension
- Agents to decrease stomach-acid secretion (such as famotidine, ranitidine, cimetidine, omeprazole)—to prevent irritation of the esophageal lining by backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux)
- Drugs that improve the propulsion of contents through the stomach and intestines (known as “gastrointestinal prokinetic agents,” such as cisapride, metoclopramide)—may increase tone of the muscle between the stomach and esophagus (gastroesophageal sphincter)
- Lidocaine solution—to manage severe esophageal pain



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat barium contrast X-rays or endoscopy every 2 to 4 weeks until clinical signs have resolved and adequate esophageal lumen size has been achieved

PREVENTIONS AND AVOIDANCE

- Proper patient preparation prior to anesthesia (12-hour preoperative fast)
- Avoid certain drugs (such as diazepam, atropine, pentobarbital, morphine, phenothiazine-derivative tranquilizers) prior to anesthesia, if possible
- If gastroesophageal reflux is present, avoid late-night feedings as they tend to decrease the ability of the muscle between the stomach and esophagus to remain closed during sleep
- Prevent animal from ingesting caustic substances and foreign bodies

POSSIBLE COMPLICATIONS

- Esophageal tear or perforation—a life-threatening complication of esophageal stricture dilation; usually occurs at the time of dilation, although it has been observed several days to weeks later
- Risk for aspiration pneumonia
- Excessive esophageal bleeding and/or introduction of bacteria into the blood stream (known as “bacteremia”) can occur secondary to esophageal dilation

EXPECTED COURSE AND PROGNOSIS

- Generally, the longer the stricture, the more guarded the prognosis
- Esophageal strictures due to scarring—generally, fair to guarded prognosis; many recur despite repeated esophageal dilation; improvement without cure is a more realistic goal
- Esophageal strictures secondary to cancer—poor prognosis



KEY POINTS

- Animals generally do not recover from untreated esophageal stricture
- Benign strictures are best treated by esophageal dilation
- Patients with esophageal strictures secondary to cancer have a poor prognosis
- High probability of happening again (recurrence) and common need for multiple dilation procedures
- Possibility of improvement (such as decreased to absent regurgitation, ability to eat softened canned foods but not dry food), but not cure

INFLAMMATION OF THE ESOPHAGUS (ESOPHAGITIS)



BASICS

OVERVIEW

- Inflammation of the esophagus—typically involves the tubular area of the esophagus itself (known as the “esophageal body”) and the muscular area between the stomach and esophagus (known as the “gastroesophageal sphincter”); occasionally the muscular area between the throat or pharynx and the esophagus (known as the “cricopharyngeal sphincter”) may be involved
- Varies from mild inflammation of the superficial lining (known as “mucosa”) of the esophagus to severe ulceration, involving underlying layers including the muscle (known as “submucosa” [that is, “layer under the mucosa”] and “muscularis” [that is, “muscle”])

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Any age; young animals with congenital (present at birth) esophageal hiatal hernia (in which a portion of the stomach enters the chest through the area where the esophagus normally passes through the diaphragm) may be at higher risk for inflammation (known as “reflux esophagitis”) secondary to backward or reverse flow of stomach contents into the esophagus
- Older animals at great risk of developing backward or reverse flow of stomach contents into the esophagus (known as “gastroesophageal reflux”) during anesthesia and then possibly inflammation of the esophagus (esophagitis)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth)
- Drooling or excessive salivation
- Howling, crying, or yelping during swallowing when the animal has active inflammation of the esophagus (known as “esophagitis”)
- Extension of the head and neck during swallowing
- Difficulty swallowing (known as “dysphagia”)
- Decreased appetite
- Weight loss
- Coughing in animals that have aspiration pneumonia
- General weakness (debilitation) with severe inflammation of the esophagus
- Inflammation and/or ulceration of the mouth and/or throat, if caustic or irritating substances have been ingested
- Fever and excessive salivation in some patients with severe ulcerative inflammation of the esophagus
- Pain on feeling or palpating the neck and esophagus
- Weight loss to severe weight loss with muscle wasting (known as “cachexia”) in patients with prolonged disease
- Abnormal lung or breathing sounds (such as wheezes and coughing)—may be detected in animals with aspiration pneumonia

CAUSES

- Backward or reverse flow of stomach contents and/or intestinal fluids into the esophagus (known as “gastroesophageal reflux”)
- Anesthesia, resulting in gastroesophageal reflux
- Ingestion of chemical irritants
- Infectious agents—calicivirus; *Pythium*, a water mold that causes pythiosis; *Candida*, a yeast that causes candidiasis or “yeast infection”
- Esophageal and/or chest surgery
- Use of feeding tube, which may irritate lining of esophagus
- Chronic vomiting
- Pills or capsules remaining in the esophagus (that is, the pill or capsule does not move through the esophagus or moves through very slowly)
- Foreign body in the esophagus

RISK FACTORS

- Hiatal hernia (in which a portion of the stomach enters the chest through the area where the esophagus normally passes through the diaphragm)—increases risk for backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux)

- Anesthesia—use of certain drugs (such as diazepam, atropine, pentobarbital, and phenothiazine-derivative tranquilizers) prior to anesthesia decreases the pressure of the muscle between the stomach and esophagus (known as the “gastroesophageal sphincter”) and can result in backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux)
- Fasting for prolonged periods or not being fasted adequately puts patients at greater risk for backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux) with anesthesia and possible inflammation of the esophagus (esophagitis)
- Inflammation of the esophagus caused by *Pythium*—usually regionally distributed in states that border the Gulf of Mexico



TREATMENT

HEALTH CARE

- Mildly affected animals can be managed as outpatients; those with more severe inflammation of the esophagus (with signs such as total lack of appetite [anorexia], dehydration, and aspiration pneumonia) require hospitalization
- Intravenous fluids to maintain hydration, as needed
- Medications—give by injection during hospitalization
- Oxygen therapy—may be necessary in patients with severe aspiration pneumonia

DIET

- Severe inflammation of the esophagus—withhold food and water for 3 to 5 days; maintain with tube feedings
- When feeding by mouth is resumed, feed small amounts in multiple feedings
- Highly digestible diet of liquid or soft consistency, with low-to-moderate fat (because high dietary fat delays emptying of the stomach) and low-fiber content

SURGERY

- Feeding tube placement using an endoscope (a lighted medical instrument that is passed into the esophagus and stomach through the mouth) or surgery is indicated in severe cases



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Usually given by injection (except for sucralfate) in severe cases
- When administering medications by mouth is resumed, dissolve medications in water and give by syringe or give directly via a feeding tube to ensure that they reach the stomach
- Sucralfate suspension
- Antibiotics—indicated for animals with aspiration pneumonia or severe ulceration or tearing (perforation) of the esophagus
- Agents to decrease stomach-acid secretion (such as famotidine, ranitidine, cimetidine, omeprazole)—to prevent occurrence of further irritation of the lining of the esophagus by backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux)
- Lidocaine solution—to manage severe esophageal pain
- Anti-inflammatory dosage of corticosteroids (such as prednisone)—to decrease the possibility of narrowing of the esophagus (esophageal stricture formation) in severe cases
- Drugs that improve the propulsion of contents through the stomach and intestines (known as “gastrointestinal prokinetic agents,” such as cisapride, metoclopramide)—may help decrease backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux)
- Fentanyl patches to relieve pain—may be useful in severe cases of painful inflammation of the esophagus
- Analogues of erythromycin—may be useful in treating cats with inflammation of the esophagus secondary to gastroesophageal reflux disease



FOLLOW-UP CARE

PATIENT MONITORING

- Patients with mild esophagitis may not require follow-up evaluation of the esophagus using an endoscope; tracking of clinical signs may be sufficient
- Consider follow-up evaluation of the esophagus using an endoscope in patients with ulcerative inflammation of the esophagus and those at risk for narrowing of the esophagus (that is, esophageal stricture)

PREVENTIONS AND AVOIDANCE

- Prevent animals from ingesting caustic substances and foreign bodies
- If backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux) is the cause of inflammation of the esophagus (esophagitis), avoid late-night feedings as they tend to decrease the ability of the muscle between the stomach and esophagus to remain closed during sleep
- Proper patient preparation (fasting) prior to anesthesia decreases the risk of gastroesophageal reflux
- Follow administration of capsules and tablets by mouth with a teaspoon (5 ml) of water in cats and dogs

POSSIBLE COMPLICATIONS

- Narrowing of the esophagus (stricture formation)
- Tearing of the esophagus (esophageal perforation)
- Aspiration pneumonia
- Permanent problems with normal function/movement of the esophagus (known as “esophageal motility dysfunction”)
- Chronic backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux) leading to inflammation of the esophagus (known as “chronic reflux esophagitis”)
- Barrett’s esophagus—rare complication of chronic reflux esophagitis characterized by ulceration of the lower esophagus, may be related to cancer of the esophagus

EXPECTED COURSE AND PROGNOSIS

- Best results when patients are treated with a diffusion barrier (such as sucralfate) and agents to decrease stomach-acid secretion (such as famotidine, ranitidine, cimetidine, omeprazole)
- Mild inflammation of the esophagus—generally favorable prognosis
- Severe or ulcerative inflammation of the esophagus—guarded prognosis
- Complete recovery is possible if the disorder is recognized and treated before serious complications develop



KEY POINTS

- Inflammation of the esophagus
- Varies from mild inflammation of the superficial lining (known as “mucosa”) of the esophagus to severe ulceration, involving underlying layers including the muscle (known as “submucosa” [that is, “layer under the mucosa”] and “muscularis” [that is, “muscle”])
- Restrict food intake by mouth in patients with severe inflammation of the esophagus to allow healing
- Potential complications include aspiration pneumonia; scarring or narrowing of the esophagus (esophageal stricture); tear in the esophagus (esophageal perforation); and/or problems with normal function/movement of the esophagus (known as “esophageal motility dysfunction”)

ETHYLENE GLYCOL (ANTIFREEZE) POISONING



BASICS

OVERVIEW

- Results from ingesting substances containing ethylene glycol (such as antifreeze)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs, cats, and many other species, including birds

Mean Age and Range

- Any age is susceptible
- Mean—3 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs are dependent on the amount of ethylene glycol (antifreeze) ingested
- Almost always sudden (acute)
- Signs caused by ethylene glycol itself and its toxic metabolites (frequently fatal); “metabolites” are substances produced by the body’s chemical processes as it breaks down the ethylene glycol
- Early signs—seen from 30 minutes to 12 hours after ingestion in dogs; nausea and vomiting; mild to severe depression; wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”) and knuckling; twitching muscles; short, rapid movements of the eyeball (known as “nystagmus”); head tremors; decreased withdrawal reflexes and righting ability; increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)
- Dogs—with increasing depression, patient drinks less but increased urination (polyuria) continues, resulting in dehydration; central nervous system signs lessen transiently after approximately 12 hours, but return later
- Cats—usually remain markedly depressed; do not exhibit increased thirst (polydipsia)
- Production of only small amounts of urine (known as “oliguria”) often develop in dogs 36 to 72 hours after ingestion of ethylene glycol (antifreeze) and in cats 12 to 24 hours after ingestion of ethylene glycol; lack of production of urine (known as “anuria”) is seen 72 to 96 hours after ingestion of ethylene glycol—if untreated
- May note severely low body temperature (known as “severe hypothermia”)
- Severe sluggishness (lethargy) or coma
- Seizures
- Lack of appetite (known as “anorexia”)
- Vomiting
- Oral ulcers
- Salivation or drooling
- Kidneys—often swollen and painful, particularly in cats

CAUSES

- Ingestion of ethylene glycol, the principal component (95%) of most antifreeze solutions

RISK FACTORS

- Access to ethylene glycol—widespread availability; somewhat pleasant taste; small minimum lethal dose (in other words, ingestion of a small amount can kill an animal); lack of public awareness of toxicity of ethylene glycol-containing antifreeze



TREATMENT

HEALTH CARE

- Cats—usually inpatient

- Dogs—usually outpatient if presented to veterinarian under 5 hours from the time of ingestion and treated with fomepizole; inpatient if presented to veterinarian more than 5 hours after the time of ingestion for intravenous fluids to correct dehydration, increase blood flow to the tissues, and promote elimination of urine (known as “diuresis”)
- Goals—prevent absorption of ethylene glycol into the body; increase excretion or removal of ethylene glycol from the body; prevent chemical processing (metabolism) of ethylene glycol by the body to toxic compounds
- Induction of vomiting and flushing of the stomach (known as “gastric lavage”) with administration of activated charcoal are not recommended, unless they can be performed in the first 30 minutes following ingestion, due to the rapid absorption of ethylene glycol
- Intravenous fluids—correct dehydration, increase blood flow to the tissues, and promote elimination of urine (diuresis); accompanied by administration of bicarbonate (given slowly intravenously) to correct metabolic acidosis (condition in which the pH of the body is too low)
- If the patient develops excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) and kidney failure characterized by production of small amounts of urine (oliguria) in dogs—most of the ethylene glycol has been metabolized; little benefit from treatment specifically designed for ethylene glycol poisoning; correct fluid, electrolyte, and acid–base disorders; promote elimination of urine (diuresis); medications to induce production and elimination of urine (known as “diuretics,” particularly mannitol) may help; peritoneal dialysis (a type of dialysis in which fluids are put into the abdomen and the lining of the abdomen [known as the “peritoneum”] acts as a filter to remove waste products from the blood; after a certain amount of time, the fluids and waste products are removed from the abdomen) may be useful; may need extended treatment (several weeks) before kidney function is reestablished

SURGERY

- Kidney transplantation—successfully employed in cats with ethylene glycol–induced kidney failure



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Dogs

- Fomepizole (4-methyl pyrazole; Antizol-Vet®)
- Ethanol, propylene glycol, and 1,3-butanediol—effectively inhibit ethylene glycol metabolism; may cause central nervous system depression; even in early stages of ethylene glycol (antifreeze) poisoning, requires hospitalization for approximately 3 days; constant intravenous (IV) infusion (ethanol and fluids); continuous monitoring for breathing and acid–base status

Cats

- Fomepizole (4-methyl pyrazole; Antizol-Vet®)—cats must be given a much higher dose of fomepizole than dogs
- Ethanol—use if fomepizole not available; given in an intravenous (IV) drip



FOLLOW-UP CARE

PATIENT MONITORING

- Blood work to monitor the kidneys (such as blood urea nitrogen [BUN]), acid–base status, and urine output—monitored daily for the first few days
- Monitor urine pH to determine response to treatment

PREVENTIONS AND AVOIDANCE

- Increasing awareness of the toxicity of ethylene glycol (found in most brands of antifreeze)—help prevent exposure; earlier treatment of patients
- Use new antifreeze products, containing propylene glycol (relatively nontoxic)

POSSIBLE COMPLICATIONS

- Without excess levels of urea and other nitrogenous waste products in the blood (azotemia)—usually no complications
- Urine concentrating ability—may be impaired with excess levels of urea and other nitrogenous waste products in the blood (azotemia); patients recover
- Death

EXPECTED COURSE AND PROGNOSIS

- Untreated—kidney failure characterized by the production of only small amounts of urine (oliguria) is seen in dogs at 36 to 72 hours after ingestion of ethylene glycol and in cats at 12 to 24 hours following ingestion; lack of production of urine (anuria) is seen by 72 to 96 hours following ingestion
- Dogs treated less than 5 hours following ingestion of ethylene glycol—prognosis is excellent with fomepizole treatment
- Dogs treated up to 8 hours following ingestion of ethylene glycol—most recover
- Dogs treated up to 36 hours following ingestion of ethylene glycol—may benefit from prevention of the body's chemical processing (metabolizing) of any remaining ethylene glycol
- Cats treated within 3 hours following ingestion of ethylene glycol—prognosis is good
- If a large quantity of ethylene glycol is ingested, prognosis is poor, unless treated within 4 hours of ingestion
- Patients with excess levels of urea and other nitrogenous waste products in the blood (azotemia) and kidney failure characterized by the production of only small amounts of urine (oliguria)—prognosis poor; almost all of the ethylene glycol will have been chemically processed by the body (metabolized) to toxic metabolites



KEY POINTS

- Ethylene glycol poisoning results from ingesting substances containing ethylene glycol (such as antifreeze)
- Signs are dependent on the amount of ethylene glycol ingested
- Ethylene glycol is readily available in many brands of antifreeze; it has a somewhat pleasant taste that attracts animals to ingest it; ethylene glycol has small minimum lethal dose (in other words, ingestion of a small amount can kill an animal)
- The public needs to be aware of toxicity of ethylene glycol-containing antifreeze; people should take precautions to safeguard their pets and other animals from potential sources of ethylene glycol

EXOCRINE PANCREATIC INSUFFICIENCY



BASICS

OVERVIEW

- The pancreas is an organ of the body, located near the upper small intestine; the pancreas produces insulin to regulate blood sugar and produces digestive enzymes involved in digestion of starches, fats, and proteins in the animal's diet; the digestive enzymes are delivered to the upper small intestine through the pancreatic duct
- Pancreatic acinar cells produce the digestive enzymes
- "Exocrine" refers to an organ or gland that secretes its products through a duct; "pancreatic" refers to the pancreas; "insufficiency" is defined as being inadequate
- Exocrine pancreatic insufficiency is a syndrome caused by inadequate production and secretion of digestive enzymes by the exocrine pancreas
- Also known as "EPI"

GENETICS

- Assumed to be hereditary in the German shepherd dog and transmitted as an autosomal recessive trait

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- German shepherd dogs

Mean Age and Range

- Young German shepherd dogs (age range approximately 1 to 4 years) with chronic diarrhea
- Wasting away or decrease in size of the cells in the pancreas that produce the digestive enzymes (known as "pancreatic acinar atrophy") in young dogs
- Long-term (chronic) inflammation of the pancreas (known as "pancreatitis") in dogs and cats of any age, but more common in middle-aged to older animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Severity—varies
- Weight loss with a normal to increased appetite; thin body
- Long-term (chronic) loose stool or diarrhea
- Diarrhea—often resembles cow feces; diarrhea may be continuous or intermittent
- Stool volumes larger than normal, with the presence of large amounts of fat in the stool, due to the inability to digest the fat (known as "steatorrhea")
- Excessive gas formation in the stomach or intestines (known as "flatulence") and rumbling or gurgling sounds caused by movement of gas in the intestinal tract (known as "borborygmus") are common, especially in dogs
- May eat feces or bowel movement (known as "coprophagia") and/or eat nonfood items (known as "pica")
- May be accompanied by increased urination (known as "polyuria") and increased thirst (known as "polydipsia"), if animal also has coexistent diabetes mellitus ("sugar diabetes") as a complication from long-term (chronic) inflammation of the pancreas (pancreatitis)
- Decreased muscle mass
- Poor-quality hair coat
- Cats with large amounts of fat in the stool, due to the inability to digest the fat (steatorrhea) may have greasy "soiling" of the hair coat around the rectum

CAUSES

- Wasting away or decrease in size of the cells in the pancreas that produce the digestive enzymes (pancreatic acinar atrophy)
- Long-term (chronic) inflammation of the pancreas (pancreatitis)
- Cancer of the pancreas (known as "pancreatic adenocarcinoma")
- Parasite—pancreatic fluke (*Eurytrema procyonis*) infestation in cats

RISK FACTORS

- Breed—German shepherd dogs
- Any condition increasing the likelihood of developing long-term (chronic) inflammation of the pancreas (pancreatitis)



TREATMENT

HEALTH CARE

- Outpatient medical management
- Patients with coexistent diabetes mellitus initially may require hospitalization for insulin regulation of high blood sugar (known as “hyperglycemia”)

DIET

- Supplementation of the diet with pancreatic enzyme replacement is the mainstay of treatment
- Type of diet does not play a role in the management of exocrine pancreatic insufficiency (EPI) in dogs and cats; however, high-fat and high-fiber diets should be avoided
- Approximately 40% of all dogs with exocrine pancreatic insufficiency (EPI) and virtually all cats with EPI are cobalamin (vitamin B12) deficient and require cobalamin supplementation
- Severely malnourished dogs also may require supplementation with tocopherol (vitamin E), and fat-soluble vitamins A, D, and K



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Use of powdered (non-enteric coated) pancreatic enzyme concentrates is the treatment of choice
- Initially—mix pancreatic enzyme powder in food at a dosage prescribed by your pet’s veterinarian with each meal; feed two meals daily to promote weight gain
- Allowing the pancreatic enzyme powder to “work” on the food several minutes prior to feeding (known as “preincubation”) does *not* improve the effectiveness of treatment
- Cobalamin (vitamin B12) supplementation is crucial, if the patient is cobalamin deficient
- Administration of antacids (such as famotidine, ranitidine, or omeprazole) may improve the condition in patients that do not respond to pancreatic enzyme treatment
- Most dogs respond to pancreatic enzyme treatment within 5 to 7 days; after a complete response has been achieved, the amount of the pancreatic enzyme supplement gradually may be reduced to a dose that prevents return of clinical signs
- Antibiotic therapy (tylosin) administered by mouth may be required for 4 to 6 weeks in dogs with coexistent small intestinal bacterial overgrowth (“SIBO”), a condition in which a high number of bacteria are found in the upper small intestine; however, SIBO resolves spontaneously in most dogs upon commencement of pancreatic enzyme replacement therapy
- The cost of pancreatic enzyme replacement is very high; some cats refuse to consume the pancreatic enzyme supplement—these patients often can be managed successfully by administration of raw beef, pork, or game pancreas; your pet’s veterinarian can provide information regarding amount to be fed (raw pancreas can be kept frozen for months without losing enzymatic activity)



FOLLOW-UP CARE

PATIENT MONITORING

- Weekly for first month of therapy
- Diarrhea improves markedly—stool consistency typically normalizes within 1 week of starting pancreatic enzyme replacement treatment
- Monitor body weight; should gain weight with treatment

- Dogs that fail to respond after 1 week of pancreatic enzyme replacement treatment should be placed on antibiotics for coexistent small intestinal bacterial overgrowth (SIBO); a condition in which a high number of bacteria are found in the upper small intestine
- Once body weight and conditioning normalize, gradually reduce the daily dosage of enzyme supplements to a level that maintains normal body weight
- Recheck serum cobalamin (vitamin B12) concentration a month after the last dose of cobalamin

PREVENTIONS AND AVOIDANCE

- Do not breed animals with wasting away or decrease in size of the cells in the pancreas that produce the digestive enzymes (pancreatic acinar atrophy)

POSSIBLE COMPLICATIONS

- 20% of dogs fail to respond to pancreatic enzymes and need further treatment
- Many patients with exocrine pancreatic insufficiency (EPI) have cobalamin (vitamin B12) deficiency and need to be managed accordingly
- Some dogs treated with pancreatic enzyme supplements develop ulcers in their mouths; in most of these dogs, the dose of pancreatic enzyme supplements can be decreased, while maintaining therapeutic response

EXPECTED COURSE AND PROGNOSIS

- Most causes are irreversible, and lifelong treatment will be required
- Dogs with exocrine pancreatic insufficiency (EPI) alone have a good prognosis with appropriate pancreatic enzyme supplementation and supportive management
- Prognosis is more guarded in patients with coexistent exocrine pancreatic insufficiency (EPI) and diabetes mellitus due to long-term (chronic) inflammation of the pancreas (pancreatitis)



KEY POINTS

- Exocrine pancreatic insufficiency (EPI) probably is inherited in German shepherd dogs; affected dogs should not be used for breeding
- Pancreatic enzymes are expensive and the animal will need lifelong treatment in most cases
- Coexistent diabetes mellitus is possible in patients with long-term (chronic) inflammation of the pancreas (pancreatitis)

EYE ABNORMALITIES PRESENT AT BIRTH



BASICS

OVERVIEW

- Single or multiple abnormalities that affect the eyeball (known as the “globe”) or the tissues surrounding the eye (known as “adnexa,” such as eyelids, third eyelid, and tear glands) that may be observed in young dogs and cats at birth or within the first 6 to 8 weeks of life
- Congenital refers to “present at birth;” congenital abnormalities may be genetic or may be caused by a problem during development of the puppy or kitten prior to birth or during birth
- The “cornea” is the clear outer layer of the front of the eye; the pupil is the circular or elliptical opening in the center of the iris of the eye; light passes through the pupil to reach the back part of the eye (known as the “retina”); the iris is the colored or pigmented part of the eye—it can be brown, blue, green, or a mixture of colors; the “lens” is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye (retina); the “retina” contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision

GENETICS

- Suspected genetic background for several congenital (present at birth) eye abnormalities, some with a unknown mode of inheritance
- Remaining strands of iris tissue (the colored or pigmented part of the eye) that may extend from one part of the iris to another or from the iris to the lining of the cornea (the clear outer layer of the front of the eye) or from the iris to the lens (the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye [retina]); condition known as “persistent pupillary membranes”—simple autosomal dominant trait in Basenjis
- Inherited embryonic developmental abnormalities of the eye (known as “persistent hyperplastic tunica vasculosa lentis” or “PHTVL” and “persistent hyperplastic primary vitreous” or “PHPV”)—autosomal dominant allele with variable expression in Doberman Pinschers; inherited in Staffordshire bull terriers
- Abnormal development of the back part of the eye (retina; condition known as “retinal dysplasia”) in English springer spaniels—simple autosomal recessive trait
- Inherited abnormal development of the eye, leading to changes in various parts of the eye in collies (known as “collie eye anomaly”)—autosomal recessive trait
- Abnormal development of the back part of the eye (retina; condition known as “retinal dysplasia”) in briards—simple autosomal recessive trait
- Abnormal development of the light-sensitive cells in the back part of the eye (retina; condition known as “photoreceptor dysplasia”) in Collies, Irish setters, and Cardigan Welsh corgis—autosomal recessive trait
- Other types of abnormal development of the light-sensitive cells in the back part of the eye (photoreceptor dysplasia) in dogs—recessively inherited
- Abnormal development of the light-sensitive cells in the back part of the eye (photoreceptor dysplasia) in Abyssinians, domestic shorthairs, and Persians—postulated autosomal dominant trait in Abyssinians and domestic shorthairs, but autosomal recessive trait in Persians

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- See **GENETICS** and **SIGNS** for breeds commonly affected

Mean Age and Range

- Observed in young dogs and cats at birth or within the first 6 to 8 weeks of life

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on defect
- May cause no signs of disease and may be an incidental finding in a complete eye examination
- May not affect vision or may cause severe visual loss or blindness
- Small eye (known as “microphthalmos”)—a congenitally small eye (present at birth); size of the eyeball varies, but smaller than normal; easily noted by comparing the size of the eyes; more difficult to detect if both eyes (bilateral) are involved; often associated with other genetic defects, such as opacities of the cornea (the normally clear front part of the eye); persistent strands of iris tissue (persistent pupillary membrane); cataract (opacity in the normally clear lens—the lens is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye [retina]); separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment), and abnormal development of the back part of the eye (retinal dysplasia)
- Absence of the eyeball (known as “anophthalmos”)—congenital (present at birth) lack of the eyeball or globe; often associated with other genetic defects, such as opacities of the cornea (the normally clear front part of the eye); persistent strands of iris tissue (persistent pupillary membrane); cataract (opacity in the normally clear lens—the lens is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye [retina]); separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment), and abnormal development of the back part of the eye (retinal dysplasia)
- “Hidden” eyeball (known as “cryptophthalmos”)—a small eyeball or globe that is concealed by other defects in the tissues surrounding

the eye (adnexa); often associated with other genetic defects, such as opacities of the cornea (the normally clear front part of the eye); persistent strands of iris tissue (persistent pupillary membrane); cataract (opacity in the normally clear lens—the lens is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye [retina]); separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment), and abnormal development of the back part of the eye (retinal dysplasia)

- Absence of the eyelid (known as “eyelid agenesis”) or failure of the eyelid to form properly (known as a “coloboma”)—often result in congenitally open eyelids; considered hereditary in Burmese cats; usually affect the upper eyelid; may note squinting or spasmodic blinking (known as “blepharospasm”) and tearing (known as “epiphora”)
- Dermoids—congenital (present at birth), tumor-like, islands of displaced skin tissue involving either eyelids, conjunctiva, or cornea; sometimes affect more than one structure; may note squinting or spasmodic blinking (blepharospasm) and tearing epiphora)
- Congenital (present at birth) absence of central canal in the tear drainage system, through which the tears drain (known as “atresia”) and lack of normal openings on the eyelids into the tear drainage system (known as “imperforate puncta”)—affects cats and dogs; imperforate puncta: common in several dog breeds (such as cocker spaniels); results in tear streaking on the face, just below the corner of the eye closest to the nose and on the side of the nose; usually not associated with other eye abnormalities
- Congenital (present at birth) dry eye (known as “keratoconjunctivitis sicca” of “KCS”)—may occur sporadically in any dog or cat breed; may be hereditary in Yorkshire terriers; usually only one eye (unilateral) is affected; affected eye often appears smaller than the normal eye; results in a thick mucoid discharge from a red and irritated eye
- Remaining strands of iris tissue (the colored or pigmented part of the eye) that may extend from one part of the iris to another or from the iris to the lining of the cornea (the clear outer layer of the front of the eye) or from the iris to the lens (the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye [retina]); may coexist with a variety of other eye defects (such as iris defects and cataracts); affects any species; recorded in numerous dog breeds; shown to be hereditary in Basenjis
- Iris cysts—circular, pigmented or nonpigmented ball-like structures that float freely in the front part of the eye, between the cornea and the iris (known as the “anterior chamber”) or are attached to the iris or to the lining of the cornea
- Congenital (present at birth) increased pressure within the eye (glaucoma) with secondary enlargement of the eye (known as “buphthalmos”)—affects dogs and cats; rare; often note increased tearing and an enlarged, red, and painful eye
- Congenital (present at birth) abnormalities of the pupil (the center of the iris)—more than one pupil (known as “polycoria”); absence of a pupil (known as “acoria”); lack of the iris (known as “aniridia”); abnormally shaped pupil (known as “dyscoria”)
- Congenital (present at birth) cataract (opacity in the normally clear lens—the lens is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye [retina])—primary, often inherited (such as in the Cavalier King Charles spaniel) or secondary to other developmental defects; often associated with other congenital abnormalities of the lens
- Abnormal development of the back part of the eye (retina; condition known as “retinal dysplasia”)—affects a variety of dog breeds; occurs sporadically in cats; abnormalities seen with an ophthalmoscope range from localized folds of retinal tissue to localized or complete separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)
- Abnormal development of the light-sensitive cells (rods and cone) in the back part of the eye (retina; conditions known as “rod and cone dysplasias”) of dogs—rod and cone dysplasia affects Irish Setters and collies; rod dysplasia and early rod degeneration affect the Norwegian elkhound; cone degeneration affects Alaskan malamutes
- Abnormal development of the light-sensitive cells (rods and cone) in the back part of the eye (retina; conditions known as “rod and cone dysplasias”) of cats—affects Persians, Abyssinians, and American mixed-breeds; may show dilated pupils at 2 to 3 weeks of age and short, rapid movements of the eyeball (known as “nyctagmus”) at 4 to 5 weeks of age, with signs of deterioration of the back part of the eye (retina) seen with an ophthalmoscope at 8 weeks of age, and night and day blindness some weeks later
- Abnormal development of the back part of the eye (retinal dysplasia) in briards—causes night blindness, short, rapid movements of the eyeball (nyctagmus), and abnormally large pupils
- Separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment”)—seen in conjunction with inherited eye diseases (such as abnormal development of the back part of the eye [retinal dysplasia]); mainly found in Labrador retrievers, Bedlington terriers, and Sealyham terriers and with collie eye anomaly; may note a widely dilated pupil that is unresponsive to light stimuli; retinal detachment may be seen in the pupil as a funnel-shaped “curtain;” complete detachment results in blindness
- Lack of development of the optic nerve (the nerve that runs from the back of the eye to the brain; condition known as “optic nerve hypoplasia”)—occurs sporadically as a congenital (present at birth) eye defect in dogs and cats; believed to have a hereditary background in miniature and toy poodles; often results in blindness

CAUSES

- Genetic
- Spontaneous malformations of the eyeball and/or surrounding tissues
- Infections and inflammations during pregnancy—congenital (present at birth) cataract (opacity in the normally clear lens—the lens is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye [retina]); medical syndromes with multiple birth defects
- Toxicity during pregnancy
- Nutritional deficiencies



TREATMENT

HEALTH CARE

- Patients may be referred to a veterinary eye doctor (ophthalmologist) for a complete evaluation, especially if the eye abnormality affects the internal structures of the eye or vision is impaired
- No medical treatment for most congenital (present at birth) abnormalities of the eye, except possibly symptomatic treatment (such as for treatment of congenital “dry eye” [KCS])
- Inhibit self-mutilation after surgical procedures by using an Elizabethan collar or by directly bandaging the paws or eye

ACTIVITY

- Usually no change in activity is necessary

SURGERY

- Depends on specific abnormality of the eye or surrounding tissues (adnexa)
- If possible, wait to have surgery until the patient has reached adult size to avoid “overcorrecting” the defect; however, the surgery may be necessary prior to adulthood, if the abnormalities are too severe and/or may lead to complications
- Abnormalities of the tissues surrounding the eye (adnexa), such as dermoids or severe malformations of the eyelids—surgery as soon as possible
- Imperforate puncta (lack of normal openings on the eyelids into the tear drainage system)—surgically correct as soon as anesthesia is safe
- Congenital (present at birth) “dry eye” (KCS)—surgically move the duct from the parotid salivary gland to the eye (procedure known as a “parotid duct transposition”); the saliva then acts as “tears” in the eye
- Surgical removal of cataracts—congenital (present at birth) opacities in the lens (cataracts) may be associated with other eye abnormalities that may cause surgical complications
- Congenital (present at birth) increased pressure within the eye (glaucoma)—surgical removal of the eye (known as “enucleation”) or surgical removal of the contents of the eyeball with insertion of a silicone ball or prosthesis (known as an “intracocular prosthesis”) to allow the eye to have a fairly normal appearance; technique relieves pain in the eye, but the eye is still blind—usually treatments of choice; consider euthanasia if both eyes are involved (bilateral glaucoma)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Congenital (present at birth) “dry eye” (KCS)—tear substitutes (Tears Naturale® and Viscotears®), possibly in combination with antibiotics (drops or gel); cyclosporine ophthalmic (Optimmune®) ointment
- Congenital (present at birth) opacities in the lens (cataracts)—medications to dilate the pupil may be used to increase visual capability, if the cataract only involves the center of the lens



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on defect
- Congenital (present at birth) “dry eye” (KCS)—requires frequent monitoring of tear production and the status of the external eye structures
- Congenital (present at birth) opacities in the lens (cataracts) and severe persistent hyperplastic tunica vasculosa lentis (PHTVL) and persistent hyperplastic primary vitreous (PHPV)—regular checkups, usually on a 6-month basis, to monitor possible progression of abnormalities
- Large defects of the back of the eye (retina) and abnormal development of the back part of the eye (retinal dysplasia)—yearly checkups to monitor possible complete separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)

PREVENTIONS AND AVOIDANCE

- Depend on type and severity of defect
- Restrict breeding of affected animals and of known carriers of documented hereditary defects; DNA-based tests are available for many inherited defects to identify carriers

POSSIBLE COMPLICATIONS

- Depend on defect
- Untreated eyelid abnormalities (such as lack of formation of the eyelid [agenesis] or masses composed of “displaced” skin, frequently with long hair [dermoids]) and congenital (present at birth) “dry eye” (KCS)—recurrent problems with inflammation of the moist tissues

surrounding the eye (known as “conjunctivitis”) and inflammation of the cornea (known as “keratitis”)

- Congenital (present at birth) increased pressure within the eye (glaucoma)—painful, blind eye in conjunction with enlargement of the eyeball (buphthalmos); often a dry, pigmented cornea
- Abnormalities of the optic nerve on the back of the eye (retina)—may cause separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)
- Separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)—may cause bleeding within the eye, especially within the vitreous; the “vitreous” is the clear, gel-like material that fills the back part of the eye (between the lens and the retina)

EXPECTED COURSE AND PROGNOSIS

- Depend on defect and type of medical and/or surgical treatment provided
- Abnormalities of the tissues around the eye (adnexa), such as the eyelids—good prognosis with surgical treatment
- Congenital (present at birth) “dry eye” (KCS)—rather poor prognosis with medical treatment only; somewhat better prognosis with surgical treatment (parotid duct transposition)
- Congenital (present at birth) opacities in the lens (cataracts)—usually good prognosis with surgical treatment, if other structures within the eye are normal



KEY POINTS

- Congenital (present at birth) eye abnormalities can affect vision; they may progress; and complications can develop
- Blind animals may need direct supervision when exposed to a potentially hazardous environment
- Congenital (present at birth) “dry eye” (KCS)—consider medical treatment versus surgical intervention; if the eye is medicated on a regular basis, the patient may do fine, especially if some tear production exists; if client cannot medicate on a regular basis, surgery (parotid duct transposition) should be considered

FACIAL NERVE PARESIS/PARALYSIS



BASICS

OVERVIEW

- Dysfunction of the facial nerve, causing weakness (known as “paresis”) or paralysis of the muscles of the ears, eyelids, lips, and/or nostrils
- The facial nerve is the seventh cranial nerve (known as cranial nerve VII); the cranial nerves are the nerves that originate in the brain

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Paralysis of unknown cause (so called “idiopathic paralysis”)—cocker spaniels, Pembroke Welsh corgis, boxers, English setters, and domestic longhair cats

Mean Age and Range

- Adults

SIGNS/OBSERVED CHANGES in the ANIMAL

- Messy eating; food left around mouth; food falling from the side of mouth
- Excessive drooling
- Lack of symmetry of the face
- Eye—inability to close the eyelids; may have discharge containing mucus and/or pus; may have inflammation of the moist lining of the eye (known as “conjunctivitis”) or inflammation of the cornea (known as “keratitis”)
- Drooping of the ear and lip on the same side of the head
- Collapse of the nostril
- Decreased or absent reflexes of the eyes and eyelids (menace response and palpebral reflex)
- Long-term (chronic) facial nerve paresis/paralysis—patient may have deviation of the face toward the affected side due to scarring of the muscles of the face (known as “muscle fibrosis”)
- Occasionally spasms may be observed in half of the face; these patients have a “grinning” appearance to one side of the face—at times the face will appear normal, only to begin “grinning” appearance again
- When secondary to brainstem disease—altered mentation (such as drowsiness or sleepiness [known as “somnia”] or stupor); other cranial nerve and gait abnormalities may be noted

CAUSES

One-Sided (Unilateral) Peripheral Facial Nerve Weakness or Paralysis

- Unknown cause (so called “idiopathic disease”)
- Metabolic disease—inadequate levels of thyroid hormone (known as “hypothyroidism”)
- Inflammatory disease—inflammation of the middle ear or inner ear (known as “otitis media-interna”) in dogs and cats; inflammatory masses that develop from the middle ear or eustachian tube (known as “nasopharyngeal polyps”) in cats
- Tumors or cancer
- Trauma—fracture of the skull near the ear; injury to the facial nerve as it leaves the skull, near the ear
- Secondary to surgical removal of the external ear canal (so called “iatrogenic disease”)

Two-Sided (Bilateral) Peripheral Facial Nerve Weakness or Paralysis

- Unknown cause (so called “idiopathic disease”)—rare
- Inflammatory and immune-mediated disease—inflammation of several nerve roots and nerves (known as “polyradiculoneuritis”), including coonhound paralysis; diseases involving a number of nerves (known as “polyneuropathies”), myasthenia gravis (a disorder of neuromuscular transmission characterized by muscular weakness and excessive fatigue)
- Metabolic disorder—disease involving multiple nerves related to the presence of cancer somewhere in the body (known as “paraneoplastic polyneuropathy”)
- Toxic disorder—botulism
- Tumor of the pituitary gland

- Infectious disease—Lyme disease (borreliosis) in people; not proven in dogs at this time

Central Nervous System Problems that Lead to Facial Nerve Weakness or Paralysis

- Most are one-sided (unilateral) facial nerve weakness (paresis) or paralysis
- Inflammatory disease—infectious disease (such as viral, bacterial, fungal, rickettsial, or protozoal infection) and noninfectious disease (such as inflammation of the brain and spinal cord and the membranes covering them [known as “meninges”] characterized by nodular, inflammatory lesions [known as “granulomatous meningoencephalomyelitis”])
- Tumor or cancer—primary brain tumor; cancer that has spread to the brain (known as “metastatic cancer”)

RISK FACTORS

- Long-term (chronic) ear disease



TREATMENT

HEALTH CARE

- Outpatient—facial paralysis of unknown cause (idiopathic facial paralysis)
- Inpatient—initial medical work up and management of generalized (systemic) or central nervous system disease

DIET

- No change required

SURGERY

- Surgery may be indicated in some cases of inflammation of the middle ear (otitis media) to drain the middle ear (procedure known as “bullae osteotomy”)—in patients with disorders of the middle ear
- Surgery may be necessary to remove inflammatory masses that develop from the middle ear or eustachian tube (nasopharyngeal polyps) in cats



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treat specific disease, if possible
- Facial weakness (paresis) or paralysis of unknown cause (idiopathic disease)—none specific; effectiveness of steroids in treatment is unknown, although used very commonly in people to treat Bell’s palsy
- Tear replacement with artificial tears—if Schirmer tear test (technique to measure watery portion of tears) value is low; if the patient has eyelid(s) turned outward, away from the eyeball (known as “ectropion”) or protrusion of the eyeball (known as “exophthalmia”)



FOLLOW-UP CARE

PATIENT MONITORING

- Reevaluate early for evidence of superficial loss of tissue on the surface of the cornea (the clear outer layer of the front of the eye), frequently with inflammation (known as a “corneal ulcer”)
- Assess monthly for reflexes of the eye and eyelids (menace response, palpebral reflex) and lip and ear movements to evaluate return of function and condition of affected eye, although damage usually is permanent

POSSIBLE COMPLICATIONS

- Dry eye (known as “keratoconjunctivitis sicca” or “KCS”)
- Superficial loss of tissue on the surface of the cornea (the clear outer layer of the front of the eye), frequently with inflammation (corneal ulcers)

- Severe deviation of the face toward the affected side due to scarring of the muscles of the face; deviation of the lips may develop

EXPECTED COURSE AND PROGNOSIS

- Depend on cause
- Facial nerve weakness (paresis) or paralysis of unknown cause (idiopathic disease)—prognosis guarded for recovery
- Improvement may take weeks or months or may never occur



KEY POINTS

- Clinical signs may be permanent, but as muscle scarring (fibrosis) develops, a natural “tuck up” reduces lack of symmetry of the face; drooling usually stops within 2 to 4 weeks
- The other side of the face can become affected
- Eye care: the cornea on the affected side may need frequent lubrication or application of artificial tears; extra care may be needed if the animal is a breed with naturally protruding eyes (exophthalmos); check regularly for corneal ulcers
- Most animals tolerate this nerve deficit well

NEONATAL MORTALITY (“FADING SYNDROME” IN NEWBORN PUPPIES OR KITTENS)



BASICS

OVERVIEW

- Death occurring from birth to 2 weeks of age
- “Neonatal” is defined as the period immediately following birth and up to the first 14 days following birth
- “Mortality” is defined as death

GENETICS

- Inbreeding—higher incidence of having identical recessive genes in the genetic make-up of an individual (known as a “homozygous recessive genotype”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Purebred or pedigree puppies and kittens—more prone to congenital (and hereditary) defects; congenital defects are conditions present at birth, they may or may not be inherited

Mean Age and Range

- Birth to 2 weeks of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Prewaning losses—typically 10% to 30% of the litter; about 65% of these losses occur during the first week of life; greater losses in a cattery or kennel should be considered abnormal
- Historical and physical examination findings rarely help determine the diagnosis, because of the limited number of ways newborns (neonates) can respond to illness
- Low birth weight, loss of weight, and/or failure to gain weight
- Decreased activity and appetite
- Weakness
- Constantly vocal or restless early, quiet and inactive later
- Tendency to remain separate from the dam and the rest of the litter
- Low body temperature (known as “hypothermia”)—normal newborn body temperature is about 35.5° C (96° F), rising to 37° to 37.8° C (99° –100° F) during the fourth week of life; low blood-glucose levels in the blood (known as “hypoglycemia”); dehydration—common and often inter-related
- Breathing difficulties (known as “respiratory distress”), diarrhea, or dark colored urine due to the presence of hemoglobin (known as “hemoglobinuria;” hemoglobin is a breakdown product of red-blood cells)—may be seen
- Gross anatomic defects—may be detectable

CAUSES

Noninfectious

- Dam-related—difficult birth (known as “dystocia”) or prolonged labor; cannibalism; failure to produce milk (lactation failure); trauma; inattention or over attention to newborn; inadequate nutrition, including taurine deficiency in kittens (taurine is a necessary amino acid)
- Environmental—any factor that discourages nursing and allows low body temperature (hypothermia), including temperature extremes, humidity extremes, inadequate sanitation, overcrowding, and stress
- Nutritional—inadequate or ineffective nursing; low blood-glucose levels (hypoglycemia); low body temperature (hypothermia)-induced digestive malfunction
- Breakdown of red-blood cells due to the presence of antibodies from the mother in the milk (condition known as “neonatal isoerythrolysis”)—queen (mother cat) with blood type B; kitten with blood type A
- Birth defects
- Gross anatomic defects—more frequently in kittens (about 10% of nonsurviving newborn kittens) than in puppies
- Gastrointestinal abnormalities—congenital (present at birth) opening in the palate (known as “cleft palate”); lack of formation of a

section of the intestine (known as “segmental intestinal agenesis”) or lack of development of normal tubular opening of the intestines (known as “intestinal atresia”)

- Abnormalities of the head and/or face (known as “craniofacial abnormalities”)—failure of midline closure, causing herniation
- Heart defects—valvular dysplasia; ventricular septal defect; atrioventricular fistula
- Respiratory defects—chest wall abnormalities; depression of the sternum and chest (known as “pectus excavatum”); inherited defect leading to lack of removal of respiratory secretions (known as “primary ciliary dyskinesia”); surfactant deficiency
- Inborn errors of metabolism—usually autosomal recessive traits

Infectious

- Viral (puppies)—canine adenovirus type 1; canine distemper virus; canine herpesvirus; canine parvovirus type 1
- Viral (kittens)—feline calicivirus; feline leukemia virus (FeLV); feline immunodeficiency virus (FIV); feline herpesvirus type 1, feline panleukopenia virus
- Bacterial—exposure to infectious bacteria mainly across the placenta, in the birth canal, via the umbilicus, gastrointestinal tract, respiratory tract, urinary tract, or skin wounds
- Generalized bacterial infection of the newborn (known as “neonatal sepsis”)—primarily from *E. coli*, β -hemolytic *streptococcus*, coagulase-positive *staphylococcus*, and gram-negative enteric organisms
- Respiratory—*Bordetella bronchiseptica*, *Pasteurella multocida*
- Gastrointestinal tract—*E. coli*; *Salmonella*; *Campylobacter*
- *Brucella canis*—puppies
- Parasitic infection—heavy infection with intestinal worms (such as *Toxocara canis*, *Toxocara cati*, *Toxascaris leonina*, *Ancylostoma caninum*, or *Ancylostoma tubaeforme*); coccidian parasites (such as *Toxoplasma*, *Neospora*, *Isospora*, *Cryptosporidium*, or *Giardia*)

RISK FACTORS

- Subnormal birth weight or failure to grow normally—kittens: minimum daily gain of 7 to 10 grams; puppies: should double in weight by 10 to 12 days; both: 5% to 10% gain per day generally acceptable
- Difficult birth (known as “dystocia”) or prolonged labor
- Inbreeding—higher incidence of having identical recessive genes in the genetic make-up of an individual (homozygous recessive genotype)
- Sire with blood type A and queen (mother cat) with blood type B (cats)



TREATMENT

HEALTH CARE

- Correct any underlying deficiencies in husbandry or breeding selection
- Warmth—slowly warm newborn (neonate) to 36° to 36.7° C (97° to 98° F) over several hours, if necessary; provide ambient temperature of 29° to 35° C (85° to 95° F) and relative humidity of 55% to 65%
- Oxygen—supplement at 30% to 40%, if necessary
- Intravenous fluids—consider administration of warmed dextrose in water (D₅W) solution if newborn has low blood-glucose levels (hypoglycemia); administer warm lactated Ringer’s solution or half-strength lactated Ringer’s and D_{2.5}W (may be administered into a vein [intravenously], into a bone/bone marrow [intraosseously], or under the skin [subcutaneously])
- Do not attempt to feed if body temperature less than 35° C (95° F) and newborn has no sucking reflex; once warmed, encourage nursing
- Breakdown of red-blood cells (neonatal isoerythrolysis) due to the presence of antibodies from the mother cat in the milk—do not allow nursing for first 24 hours after birth



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics—commonly used are penicillins (penicillin G, ampicillin, amoxicillin, amoxicillin with clavulanic acid) and first-generation cephalosporins
- Supplement—milk-replacer formula

- Vitamin K₁



FOLLOW-UP CARE

PATIENT MONITORING

- Hydration status—check daily; dryness of mouth and yellow golden urine indicate dehydration
- Body weight—monitor daily or every other day in growing newborns (neonates)
- Dam—check that nursing and care are adequate; supplement nursing with milk-replacer formula, if necessary

PREVENTIONS AND AVOIDANCE

- Breakdown of red-blood cells (neonatal isoerythrolysis) due to the presence of antibodies from the queen (mother cat) in the milk—do not allow nursing for first 24 hours after birth



KEY POINTS

- Death occurring from birth to 2 weeks of age
- Purebred or pedigree puppies and kittens—more prone to congenital (and hereditary) defects; congenital defects are conditions present at birth, they may or may not be inherited
- Body weight—monitor daily or every other day in growing newborns (neonates)
- Dam—check that nursing and care are adequate; supplement nursing with milk-replacer formula, if necessary

FALSE PREGNANCY



BASICS

OVERVIEW

- Display of maternal behavior and physical signs of pregnancy 2 to 3 months after “heat” or “estrus” by a nonpregnant bitch
- A female dog is a “bitch”
- Also known as “pseudopregnancy,” “phantom pregnancy,” or “pseudocyesis”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Common in dogs
- Rare in cats

Breed Predilection

- None

Mean Age and Range

- Any age

Predominant Sex

- Nonpregnant females that were in heat or estrus 2 to 3 months earlier and that are experiencing a decline in serum progesterone concentration; “progesterone” is the female hormone that supports and maintains pregnancy in a pregnant animal—it normally remains high in nonpregnant bitches for several weeks following their heat or estrous cycles

SIGNS/OBSERVED CHANGES in the ANIMAL

- Severity variable among individuals and from one occurrence to the next within the same individual
- Behavior changes—nesting, mothering activity (such as mothering a stuffed toy), restlessness, and self-nursing
- Abdominal distention and breast or mammary gland enlargement
- Vomiting, depression, and lack of appetite (known as “anorexia”)
- Signs of labor (rare)
- Large mammary glands that secrete a brownish fluid or milk

CAUSES

- False pregnancy is a normal phenomenon in bitches following ovulation
- Progesterone and prolactin—drop in progesterone concentration causes prolactin concentration to rise; “progesterone” is the female hormone that supports and maintains pregnancy in a pregnant animal—it normally remains high in nonpregnant bitches for several weeks following heat or estrus; “prolactin” is a hormone that stimulates breast or mammary gland enlargement and milk production (known as “lactation”)
- Treatment with progestin (substance capable of producing the effects of the female hormone, progesterone) for conditions not related to false pregnancy—may develop signs of false pregnancy after drug withdrawal
- Surgical removal of the ovaries (known as “oophorectomy” or “ovariectomy”) or the ovaries and uterus (known as “ovariohysterectomy” or “spay”) during the period when progesterone levels are high following heat or estrus—may develop signs of false pregnancy 3 to 4 days following surgery due to the sudden drop in progesterone
- Inadequate levels of thyroid hormone (known as “hypothyroidism”) with high thyroid-stimulating hormone (TSH) concentration in the blood, which stimulates prolactin secretion—may note some associated clinical signs

RISK FACTORS

- Not thought to be influenced by previous pregnancy
- Does not cause susceptibility to other reproductive diseases



TREATMENT

HEALTH CARE

- Outpatient for medical treatment
- Inpatient—planned surgery
- Treatment usually unnecessary—all pregnant, nonpregnant, and false-pregnant ovulating dogs go through a similar stage of high progesterone levels following heat or estrus
- Progestins (substances capable of producing the effects of the female hormone, progesterone) and androgens (male hormones) to decrease secretion of prolactin; “prolactin” is a hormone that stimulates breast or mammary gland enlargement and milk production (lactation)
- Surgical removal of the ovaries and uterus (ovariohysterectomy or spay) during anestrus (the time between heat or estrous cycles)—prevents recurrence
- Mammary glands—minimize stimuli that promote milk production (lactation); use cold and warm packs applied to the breasts or mammary glands
- Elizabethan collar—prevent self-nursing or licking; but even rubbing of the collar on the mammary glands may be sufficient to prolong milk production (lactation)

DIET

- Reduction of food over 3 to 4 days may reduce milk production (lactation)

SURGERY

- Surgical removal of the ovaries and uterus (ovariohysterectomy or spay)—if fertility not an issue; recommended during the next anestrus (the time between heat or estrous cycles); do not perform while pet has clinical signs of false pregnancy, because surgery will not alleviate signs and follow-up medical treatment may be required



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Bromocriptine (Parlodel®)—not approved for veterinary use in the United States and Canada; will reduce milk production (lactation) by 89% and decrease behavioral signs of false pregnancy by 90%
- Cabergoline (Dostinex®)—a prolactin inhibitor; overall success rate 95%; “prolactin” is a hormone that stimulates breast or mammary gland enlargement and milk production (lactation)
- Testosterone—may cause development of male-like characteristics in females; should not be used in animals with liver or kidney conditions or during pregnancy (causes masculinization of female fetuses)
- Mibolerone (Cheque® Drops)—same potential side effects as testosterone
- Megestrol acetate (Ovaban®)—may cause enlargement of the breasts or mammary glands; inflammation with accumulation of pus in the uterus (known as “pyometra”); diabetes mellitus (sugar diabetes); increased appetite; weight gain; and changes in the adrenal glands; signs of false pregnancy may return after discontinuation of treatment; therefore, generally not recommended
- Mild tranquilizers may reduce behavioral signs



FOLLOW-UP CARE

PREVENTIONS AND AVOIDANCE

- Surgical removal of the ovaries and uterus (ovariohysterectomy or spay) during anestrus (the time between heat or estrous cycles)—prevents recurrence

EXPECTED COURSE AND PROGNOSIS

- Usually resolves in 2 to 3 weeks without treatment
- Treatment with bromocriptine or cabergoline—may resolve condition in 1 week
- False pregnancy may develop during subsequent heat or estrous cycles



KEY POINTS

- False pregnancy is a normal phenomenon in bitches following ovulation
- No association has been identified between false pregnancy and reproductive abnormalities
- If a litter is desired, breed the bitch during the next heat or estrus

FELINE CALICIVIRUS INFECTION



BASICS

OVERVIEW

- A common viral disease of domestic and exotic cats characterized by upper respiratory tract signs, ulcers in the mouth, pneumonia, and occasionally inflammation of the joints (known as “arthritis”), or a highly fatal, generalized (systemic) disease with bleeding and fever
- “Upper respiratory tract” (also known as the “upper airways”) includes the nose, nasal passages, throat (pharynx), and windpipe (trachea)
- “Lower respiratory tract” (also known as the “lower airways”) includes the bronchi, bronchioles, and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Mean Age and Range

- Young kittens greater than 6 weeks of age—most common
- Cats of any age may show clinical disease

SIGNS/OBSERVED CHANGES in the ANIMAL

- May present as an upper respiratory infection with eye and nose involvement, as an ulcerative disease primarily of the mouth, as pneumonia, as a sudden (acute) inflammation of the joints (arthritis), as a generalized (systemic) disease characterized by bleeding, or any combination of these
- Sudden onset
- Generally alert and in good body condition
- Lack of appetite (known as “anorexia”)
- Discharge from the eyes or nose, usually with little or no sneezing
- Ulcers on the tongue, hard palate, lips, tip of nose, or around the claws; ulcers may occur without other signs
- Difficulty breathing (known as “dyspnea”) from pneumonia
- Sudden (acute), painful lameness
- Fever
- Evidence of generalized (systemic) bleeding

CAUSES

- Calicivirus
- Numerous strains exist in nature

RISK FACTORS

- Lack of vaccination or improper vaccination
- Multicat facilities
- Coexistent infections with other disease-causing agents (such as feline herpesvirus-1 or feline parvovirus)
- Poor air circulation (ventilation) in multicat facilities (such as catteries)



TREATMENT

HEALTH CARE

- Outpatient, unless severe pneumonia or bleeding occurs
- Clean eyes and nose, as needed

- Oxygen—for cases with severe pneumonia

ACTIVITY

- Patients should be restricted from contact with other cats to prevent transmission of the calicivirus

DIET

- No restrictions
- Special diets—perhaps to entice cats to resume eating
- Provide soft foods—if ulcers in the mouth restrict eating



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No specific medications to eliminate the virus (known as “antiviral drugs”) are effective
- Broad-spectrum antibiotics—may be indicated (such as amoxicillin); however, secondary bacterial infections of affected cats are not nearly as important as for cats with feline herpesvirus-1 infections
- Antibiotic eye ointments—to reduce secondary bacterial infections of the moist tissues of the eye (known as the “conjunctiva”)
- Appropriate pain medication—for transient pain from inflammation of the joints (arthritis); should be administered only under the direction of your pet’s veterinarian



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor for sudden development of difficulty breathing (dyspnea) associated with pneumonia
- No specific laboratory tests

PREVENTIONS AND AVOIDANCE

- All cats should be vaccinated at the same time they are vaccinated against feline herpesvirus-1; routine vaccination with either modified live virus (MLV) vaccine or inactivated vaccines should be done at 8 to 10 weeks of age and repeated 3 to 4 weeks later
- Breeding catteries—respiratory disease is a problem; vaccinate kittens at an earlier age, either with an additional vaccination at 4 to 5 weeks of age or with an intranasal vaccine at 10 to 14 days of age; follow-up vaccinations at 6, 10, and 14 weeks of age
- American Association of Feline Practitioners—classifies feline herpesvirus, feline parvovirus (panleukopenia), and feline calicivirus as core vaccines; vaccinate all cats with these three agents on the initial visit, after 12 weeks of age, and 1 year later; revaccinate for calicivirus every 3 years
- Vaccination will not prevent virus infection in a subsequent exposure, but will prevent serious clinical disease caused by most strains of calicivirus

POSSIBLE COMPLICATIONS

- Pneumonia—most serious complication; can be life-threatening
- Secondary bacterial infections of the lungs or upper airways
- Ulcers of the mouth and the sudden (acute) inflammation of the joints (arthritis) usually heal without complications
- Generalized (systemic) bleeding disease may be fatal

EXPECTED COURSE AND PROGNOSIS

- Clinical disease—usually appears 3 to 4 days after exposure to calicivirus
- Once antibodies appear, about 7 days after exposure, recovery is usually rapid; an “antibody” is a protein that is produced by the immune system in response to a specific antigen (in this case, the calicivirus)—when the body is exposed to the antigen, the antibody responds
- Prognosis excellent, unless severe pneumonia or generalized (systemic) bleeding disease develops
- Recovered cats—persistently infected for long periods; will shed small quantities of virus continuously in secretions from the mouth



KEY POINTS

- Proper vaccination is very important in controlling development of clinical disease for most strains of calicivirus
- Modify the vaccination protocol in breeding catteries to include kittens before they become infected (often at 6 to 8 weeks of age) from a mother cat carrying the virus (known as a “carrier queen”)
- Calicivirus is relatively stable in the environment and is resistant to many disinfectants

FELINE IDIOPATHIC LOWER URINARY TRACT DISEASE



BASICS

OVERVIEW

- “Idiopathic” refers to a disease of unknown cause; the “lower urinary tract” refers to the bladder and urethra (the tube from the bladder to the outside, through which urine flows out of the body)
- “Feline idiopathic lower urinary tract disease” is a general term for disorders of domestic cats characterized by blood in the urine (known as “hematuria”); difficult or painful urination (known as “dysuria”); abnormal, frequent passage of urine (known as “pollakiuria”); urinating in inappropriate locations (known as “periuria”), such as in a bath tub; and partial or complete blockage of the urethra (known as “urethral obstruction”)
- Varying combinations of these signs may be associated with any cause of feline lower urinary tract disease (also known as “FLUTD”)
- Similarity of clinical signs with diverse causes is not surprising, since the feline urinary tract responds to various diseases in a limited and predictable fashion
- Idiopathic feline urinary tract disease and inflammation of the bladder for unknown reasons (known as “idiopathic cystitis”) are diagnoses that are established only after known causes (such as kidney stones or urinary tract infection) of the described signs of urinary tract disease have been eliminated
- “Feline urologic syndrome” or “FUS” previously has been used by the veterinary profession as a diagnosis for the described signs of FLUTD; “feline urologic syndrome” is a misnomer as it is not a “syndrome” or a diagnosis, but rather a description of a collection of signs; when used, “FUS” should be defined as “feline urologic signs”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Male and female cats
- The incidence of blood in the urine (hematuria), difficult or painful urination (dysuria), and/or blockage of the urethra (urethral obstruction) in domestic cats in the United States and Great Britain previously has been reported to be approximately 0.5% to 1.0% per year

Mean Age and Range

- May occur at any age, but is recognized most commonly in young to middle-aged adults (mean age, 3.5 years)
- Uncommon in cats less than 1 year of age and in cats greater than 10 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Difficult or painful urination (dysuria)
- Blood in the urine (hematuria)
- Abnormal, frequent passage of urine (pollakiuria)
- Urinating in inappropriate locations (periuria)
- Blockage of urine flow through the urethra to outside the body (known as “outflow obstruction”)
- Thickened, firm, contracted bladder wall felt by the veterinarian during physical examination
- Some cats with lower urinary tract diseases exhibit findings similar to those observed in humans with interstitial cystitis

CAUSES

By definition, this is “idiopathic” disease for which the cause is unknown. Many possible causes have been suggested, such as the following:

- Noninfectious diseases, including interstitial cystitis
- Viruses implicated—calicivirus, feline syncytia-forming virus, and a gamma herpesvirus (bovine herpesvirus 4) have been suggested as potential causes in some cats
- Initial episodes of idiopathic lower urinary tract diseases usually occur in the absence of a significant number of detectable bacteria and white blood cells in the urine (known as “pyuria”); studies of male and female cats with and without blockage of the urethra (urethral obstruction) found bacterial urinary tract infections in less than 3% of young to middle-age adults and approximately 10% of senior cats

RISK FACTORS

- Stress—may play a role in causing signs or in making the signs worse; unlikely to be a primary cause of FLUTD



TREATMENT

HEALTH CARE

- Patients without blockage of the urethra (nonobstructive lower urinary tract diseases)—typically managed as outpatients; diagnostic evaluation may require brief hospitalization
- Patients with blockage of the urethra (obstructive lower urinary tract diseases)—usually hospitalized for diagnosis and management

DIET

- Appropriate dietary management recommended for cats with persistent presence of crystals in the urine (known as “crystalluria”) associated with plugs in the urethra, causing blockage of the urethra (urethral obstruction)
- Observations suggest that feeding moist rather than dry foods may minimize recurrence of signs; the goals are to promote flushing action of the bladder and urethra by increasing urine volume and to dilute concentrations of toxins, chemical irritants, and substances that lead to inflammation (known as “inflammatory mediators”), and components needed to produce urinary tract stones (known as “uroliths”)

SURGERY

- Surgical incision into the bladder (known as “cystotomy”) to flush (lavage) and remove inflamed tissue (debride) the lining of the bladder mucosa is not recommended
- Surgical opening of the urethra (known as “perineal urethrostomy”) to minimize recurrent blockage of the urethra (urethral obstruction) should be considered only when the obstructive disease is localized to the area of the urethra within the penis (known as the “penile urethra”) by contrast X-rays of the urethra



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Propantheline may be considered to minimize hyperactivity of the bladder detrusor muscle and urge incontinence or the need to urinate frequently
- Amitriptyline, a tricyclic antidepressant (TCA) and anti-anxiety drug—has been suggested as a treatment for cats with severe recurrent or persistent signs
- Butorphanol, buprenorphine, and fentanyl—have been suggested for short-term pain relief (analgesia) in cats with inflammation of the bladder for unknown reasons (idiopathic cystitis)
- Phenoxybenzamine—may be used to minimize muscular spasm in the urethra and thus minimize blockage of urine flow related to muscle spasm
- Pentosan polysulfate sodium, a semisynthetic glycosaminoglycan—suggested to help repair the glycosaminoglycan coating of the lining of the urinary tract (glycosaminoglycan is a protein-carbohydrate compound, also known as “GAG”)
- Glucosamine alone or in combination with chondroitin sulfate has been suggested to help repair the damaged GAG layer coating the lining of the urinary tract
- Steroids—no detectable effect on relieving clinical signs; may make bacterial urinary tract infection more likely, especially in cats with catheters placed in their urethras to allow removal of urine from the bladder
- Nonsteroidal anti-inflammatory drugs (NSAIDs)—suggested by some, because of their anti-inflammatory and pain-relieving (analgesic) properties
- Dimethylsulfoxide (DMSO)—no detectable effect on relieving clinical signs
- Antibiotics and methenamine—no detectable effect on relieving clinical signs



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor blood in the urine (hematuria) by urinalysis; tapping the bladder (known as “cystocentesis”) to obtain a urine sample may introduce blood into the urine (known as “iatrogenic hematuria”), so collecting a urine sample from naturally voided urine is preferred

PREVENTIONS AND AVOIDANCE

- Observations suggest that feeding moist rather than dry foods may minimize recurrence of signs
- Reduce environmental stress

POSSIBLE COMPLICATIONS

- Use of catheters placed into the urethra and bladder to allow removal of urine from the bladder (known as “indwelling transurethral catheters”)—often cause trauma; may lead to bacterial urinary tract infections, because the catheter provides a pathway for bacteria to move up the urethra and into the bladder (and possibly to the kidneys)
- Surgical opening of the urethra (known as “perineal urethrostomy”)—may allow increased likelihood of bacterial urinary tract infections; postoperative scarring may lead to narrowing of the urethra (known as “urethral stricture”)

EXPECTED COURSE AND PROGNOSIS

- Blood in the urine (hematuria), difficult or painful urination (dysuria), and abnormal frequent passage of urine (pollakiuria) often are self-limiting in patients with most idiopathic lower urinary tract diseases; signs generally subside within 4 to 7 days
- Signs often recur unpredictably; frequency of recurrence appears to decline with advancing age



KEY POINTS

- Blood in the urine (hematuria), difficult or painful urination (dysuria), and abnormal frequent passage of urine (pollakiuria) often are self-limiting in patients with most idiopathic lower urinary tract diseases; signs generally subside within 4 to 7 days
- Signs often recur unpredictably; frequency of recurrence appears to decline with advancing age
- FLUTD generally is treated symptomatically; few controlled scientific studies demonstrate effectiveness of most drugs used
- Male cats should be monitored for signs of blockage of the urethra (urethral obstruction)
- Reduce environmental stress by minimizing impact of changes in the home, and maintaining a constant diet; environmental enrichment for indoor-housed cats consists of providing necessary resources (such as food, water, litter boxes, space, play), providing a safe place to hide, refinement of cat-owner interactions, and management of conflict
- Provide proper litter-box hygiene

FELINE IMMUNODEFICIENCY VIRUS (FIV) INFECTION



BASICS

OVERVIEW

- A complex retrovirus that causes immunodeficiency disease in domestic cats
- “Immunodeficiency” is the medical term for inability to develop a normal immune response
- Feline immunodeficiency virus is in the same genus (Lentivirus) of viruses as human immunodeficiency virus (HIV), the causative agent of acquired immunodeficiency syndrome (AIDS) in people

GENETICS

- No genetic susceptibility for infection
- Genetics may play a role in progression and severity of disease

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Mean Age and Range

- Likelihood of infection increases with age
- Mean age—5 years of age at time of diagnosis

Predominant Sex

- Male—more aggressive; more likely to roam (increasing exposure to virus)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Diverse signs owing to the decreased ability to develop a normal immune response (that is, the immunosuppressive nature of infection)
- Associated disease cannot be distinguished clinically from feline leukemia virus (FeLV)-associated immunodeficiencies
- Recurrent minor illnesses, especially with upper respiratory and gastrointestinal signs
- Enlarged lymph nodes (known as “lymphadenomegaly”)—mild to moderate
- Inflammation of the gums (known as “gingivitis”), of the mouth (known as “stomatitis”), and/or of the tissues surrounding and supporting the teeth (known as “periodontitis”), seen in 25% to 50% of cases
- Upper respiratory tract disease seen in 30% of cases—inflammation of the nose (known as “rhinitis”); inflammation of the moist tissues of the eye (known as “conjunctivitis”); inflammation of the cornea (known as “keratitis”)—the cornea is the clear part of the eye, located in the front of the eyeball; often associated with feline herpesvirus and calicivirus infections
- Long-term (chronic) kidney insufficiency
- Persistent diarrhea, seen in 10% to 20% of cases
- Long-term (chronic), nonresponsive, or recurrent infections of the external ear and skin—from bacterial or fungal infections
- Fever and wasting—especially in later stage
- Eye disease—inflammation of the front part of the eye, including the iris (known as “anterior uveitis”); disease of the eye, in which the pressure within the eye is increased (known as “glaucoma”)
- Cancer (such as lymphoma; “lymphoma” is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body)
- Nervous system abnormalities—disruption of normal sleep patterns; behavioral changes (such as pacing and aggression); changes in vision and hearing; disorders usually affecting the nerves to the legs and paws (known as “peripheral neuropathies”)

CAUSES

- Cat-to-cat transmission; usually by bite wounds
- Occasional transmission of the virus at the time of birth
- Sexual transmission uncommon, although feline immunodeficiency virus (FIV) has been detected in semen

RISK FACTORS

- Male
- Free-roaming cat



TREATMENT

HEALTH CARE

- Outpatient sufficient for most patients
- Inpatient—with severe secondary infections, until condition is stable
- Primary consideration—manage secondary and opportunistic infections; “opportunistic infections” are infections caused by organisms that usually do not cause disease, but are able to cause disease because the animal’s body and/or immune system has been weakened, in this case by the feline immunodeficiency virus (FIV) infection
- Supportive treatment—fluids and nutritional supplements, as necessary

ACTIVITY

- Normal

DIET

- Normal
- Diarrhea, kidney disease, or long-term (chronic) wasting—special diet, as necessary

SURGERY

- Dental cleaning, tooth extraction, biopsy of the gums
- Biopsy or surgical removal of tumors



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Zidovudine (Retrovir®)—direct antiviral agent; most effective against sudden (acute) infection; monitor for bone-marrow toxicity
- Medications to alter the immune response (known as “immunomodulatory drugs”)—alleviate some clinical signs; may increase survival rates and improve clinical status; examples include interferon (Roferon®); feline omega-interferon (Virbagen® Omega); also try *Propionibacterium acnes* (ImmunoRegulin®), or acemannan (Carrisyn™)
- Antibiotic or antifungal drugs—useful for overgrowth of bacteria or fungi; prolonged therapy or high dosages may be required; examples include metronidazole and clindamycin
- Medications to decrease the immune response (such as steroids or gold salts)—judicious, but aggressive use may help control immune-mediated inflammation
- Short-term appetite stimulation: diazepam or oxazepam; more prolonged appetite stimulation and reversal of extreme weight loss with muscle wasting (known as “cachexia”): anabolic steroids or megestrol acetate; efficacy in feline immunodeficiency virus (FIV)-positive cats is unknown
- Steroids applied directly to the eye (topical steroids)—for inflammation of the front part of the eye, including the iris (anterior uveitis); long-term response may be incomplete or poor
- Yearly vaccination for respiratory and intestinal viruses with inactivated vaccines is recommended



FOLLOW-UP CARE

PATIENT MONITORING

- Varies according to secondary infections and other manifestations of disease

PREVENTIONS AND AVOIDANCE

- Prevent contact with feline immunodeficiency virus (FIV)-positive cats

- Quarantine and test incoming cats for FIV before introducing into households currently with one or more cats

Vaccine

- Inactivated whole virus vaccine (Fel-O-Vax® FIV, Fort Dodge Animal Health)
- Cannot distinguish between vaccinated and feline immunodeficiency virus (FIV)-infected cats with antibody assays; virus detection by polymerase chain reaction (PCR) is inconsistent—makes diagnosis of disease difficult
- Discuss the use of the vaccine with your cat's veterinarian

POSSIBLE COMPLICATIONS

- Recurrent infections
- Wasting
- Death

EXPECTED COURSE AND PROGNOSIS

- Within the first 2 years after diagnosis or 4.5 to 6 years after the estimated time of infection, about 20% of cats die, but over 50% remain with no clinical signs of disease
- In late stages of disease (wasting and frequent or severe opportunistic infections), life expectancy is less than 1 year
- Inflammation of the gums (gingivitis) and mouth (stomatitis)—may not respond to treatment or may be difficult to treat



KEY POINTS

- Feline immunodeficiency virus (FIV) infection is slowly progressive and infected cats may remain healthy for years
- Cats with clinical signs will have recurrent or long-term (chronic) health problems that require medical attention
- Keep feline immunodeficiency virus (FIV) infected cats indoors to protect them from exposure to secondary disease-causing agents and to prevent spread of FIV to other cats

FELINE INFECTIOUS PERITONITIS (FIP)



BASICS

OVERVIEW

- A generalized (systemic), viral disease characterized by subtle onset of signs, persistent fever that does not respond to treatment, inflammatory nodular tissue reaction with the presence of pus (known as “pyogranulomatous tissue reaction”), accumulation of inflammatory fluids in body cavities, and high mortality

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats—domestic and exotic

Breed Predilections

- Some families or lines of cats appear more susceptible to feline infectious peritonitis (FIP)

Mean Age and Range

- Highest incidence of disease—in kittens 3 months to 3 years of age
- Incidence of disease decreases sharply after cats reach 3 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- A wide range of signs, depending on the strain of virus, effectiveness of the cat’s immune response, and organ system affected
- Two classic forms—“wet” or “effusive” form, targets the body cavities; “dry” or “non-effusive” form, targets a variety of organs
- Subtle onset of signs
- Gradual weight loss and decrease in appetite
- Gradual increase in the size of the abdomen, giving the cat a potbellied appearance
- Persistent fever—fluctuating; antibiotic unresponsive
- Depression
- Poor condition
- Stunted growth
- Dull, rough hair coat
- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)
- Build-up of fluid in the abdomen (known as “abdominal effusion”) and/or in the space between the chest wall and lungs (known as “pleural effusion”)
- Palpation of the abdomen—abdominal masses (nodular masses with or without pus) on the surface of various organs, especially the kidney, and within the intestinal wall; lymph nodes may be enlarged
- Eyes—inflammation of the front part of the eye, including the iris (known as “anterior uveitis”); aggregates of inflammatory cells adhering to various areas of the inner lining of the cornea (known as “keratic precipitates”); color change to the iris (the pigmented part of the eye); and irregularly shaped pupil (the circular or elliptical opening in the center of the iris of the eye)
- Nervous system signs, determined by the location (for example, brain or spinal cord) involved

CAUSES

- Two types of feline coronavirus—feline coronavirus-1 (FCoV-1) that causes perhaps 85% of infections and feline coronavirus-2 (FCoV-2)

RISK FACTORS

- Contact with a feline coronavirus-positive cat
- Breeding catteries or multicat facilities
- Less than 3 years of age
- Feline leukemia virus (FeLV) infection



TREATMENT

HEALTH CARE

- No treatment routinely is effective
- Inpatient or outpatient, depending on stage and severity of disease and owner's willingness and ability to provide good supportive care
- Therapeutic tapping of a body cavity to remove fluid (known as "paracentesis")—to relieve pressure from excessive fluid build-up in the abdomen (abdominal effusion) or in the space between the chest wall and lungs (pleural effusion)
- Important to encourage the affected cat to eat

ACTIVITY

- Restrict to prevent exposure of other cats, although greatest degree of virus shed occurs before the patient shows signs

DIET

- Any food that will entice the patient to eat

SURGERY

- Generally none
- Rarely, inflammatory abdominal disease from feline coronavirus may cause intestinal blockage or obstruction; abdominal surgery may be required



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medications to decrease the immune response (known as "immunosuppressive drugs"), such as prednisolone and cyclophosphamide—limited success
- Steroids injected under the moist tissues of the eyes (known as "subconjunctival steroid injection")—may help eye involvement
- Interferon—limited success in treatment; a recombinant interferon reported to have some success in Japan
- Antibiotics—ineffective because generally not associated with secondary bacterial infections
- No antiviral drugs proven to be effective



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor for development of large quantities of fluid build-up in the space between the chest wall and lungs (pleural effusion)

PREVENTIONS AND AVOIDANCE

- Modified live virus (MLV) intranasal vaccine—available against feline coronavirus/FIP virus; effectiveness of vaccine is low; cannot rely on vaccination alone for control; may produce antibody-positive cats, complicating monitoring in catteries or colonies—talk to your cat's veterinarian about this vaccine
- Mother/offspring—main method of transmission appears to be from mother cat ("the queen") that is carrying the virus, but does not have signs of disease (known as an "asymptomatic carrier queen") to her kittens at 5 to 7 weeks of age; break cycle of transmission by early weaning at 4 to 5 weeks of age and isolating litter from direct contact with other cats, including the queen
- Routine disinfection—premise, cages, and water/food dishes; readily inactivates virus; reduces transmission
- Introduce only feline coronavirus antibody-negative cats to catteries or colonies that are free of virus
- Restrict household cats to indoor environments

POSSIBLE COMPLICATIONS

- Fluid build-up in the space between the chest wall and lungs (pleural effusion) may lead to breathing difficulties and require tapping of the chest and removal of fluid (known as “thoracocentesis”)
- Intestinal blockage or obstruction from inflammatory abdominal disease
- Central nervous system disease
- Death

EXPECTED COURSE AND PROGNOSIS

- Most feline coronavirus-positive cats have subclinical infection or mild, localized nodular inflammatory disease that is not diagnosed as feline infectious peritonitis (FIP); a “subclinical infection” is one in which the animal is infected, but has no signs of disease
- Patients with generalized and typical signs of feline infectious peritonitis almost invariably die
- Clinical course—a few days to several months
- Prognosis grave once typical signs occur; mortality nearly 100%



KEY POINTS

- Feline infectious peritonitis (FIP) has a grave prognosis
- Feline coronavirus infection is common; however, likelihood of developing actual clinical disease is low; less than 10% of feline coronavirus antibody-positive cats less than 3 years of age eventually develop clinical disease

FELINE ISCHEMIC ENCEPHALOPATHY

(BRAIN DISORDER IN CATS)



BASICS

OVERVIEW

- “Feline” refers to cat; “ischemic” refers to ischemia; “ischemia” is the loss of blood or blood flow to a tissue or organ, usually due to some type of blockage of the blood vessels; “encephalopathy” is a disorder of the brain
- “Feline ischemic encephalopathy” is a seasonal nervous system disease that occurs in outdoor cats or cats with access to the outdoors in North America during the summer months; usually results in sudden (acute) onset of seizures, circling, altered mentation and/or blindness
- Abnormal migration of a *Cuterebra* larva in the brain of a cat that often causes a blood clot in a blood vessel (known as “thrombosis”) or contraction or narrowing (known as “vasospasm”) of the middle cerebral artery with resulting loss of blood flow and death of nerve tissues in part of the brain (condition known as “ischemic necrosis”); also may involve loss of function (known as “degeneration”) of the brain as well as nerve tissue destruction associated with the actual physical migration of the *Cuterebra* larva in the brain tissue
- Must be differentiated from other causes of blood vessel (known as “vascular”) diseases affecting the brain of cats, as well as other nervous system diseases of the cat

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Mean Age and Range

- Mean, 2 years of age
- Range, 1 to 7 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden (acute) onset of nervous system signs
- Often preceded by upper respiratory signs 1 to 3 weeks prior to the nervous system signs (due to the migration of the parasite [*Cuterebra*] in the nasal passage)
- Most common signs—seizures, circling, altered mentation, blindness
- Sometimes nervous system signs can originate from multiple locations in the nervous system; rarely see spinal cord signs (such as weakness or paralysis)

CAUSES

- *Cuterebra* larvae; *Cuterebra* is a large fly, which lays eggs in the ground at the opening of rodent burrows; the eggs hatch and the larvae attach to the hair and skin of a host (such as a rabbit); they enter the nose and penetrate the moist lining, from which they migrate and eventually reach a location under the skin, where they continue to develop; the cat may become a host as it hunts or moves in the area where the larvae are located—in most cases, the larvae migrate to locations under the skin (especially around the head) of the cat, but if they follow an abnormal migration pattern in the cat’s body, they may end up in the brain

RISK FACTORS

- Outdoor cats; access to the outdoors
- July, August and September in the northeast United States and southeast Canada
- Hunting cats



TREATMENT

HEALTH CARE

- Padded cage may be necessary if the cat is having numerous seizures

SURGERY

- Surgical removal of the parasite from the brain has not been reported in cats



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Supportive care and appropriate fluid supplementation, which may include thiamine (a B vitamin) and additional potassium (administered intravenously), depending on the nutritional status of the patient
- Medications to control seizures (known as “antiepileptic drugs”), such as phenobarbital or diazepam to stop cluster seizures or repeated or prolonged seizure activity (known as “status epilepticus”)
- A “cocktail treatment” has been proposed for recently affected cats, which includes diphenhydramine administered by intramuscular (IM) injection before giving ivermectin administered by subcutaneous (SC; under the skin) injection and prednisolone sodium succinate administered by intravenous (IV) injection; in addition patients receive prednisone and an antibiotic, enrofloxacin, administered by mouth (PO); ivermectin is not approved for use against *Cuterebra* larvae, so your veterinarian will discuss the use of this medication with you prior to treating your cat
- The previously described “cocktail treatment” for feline ischemic encephalopathy is not necessary in a cat not recently showing clinical signs (that is, more than a week since having clinical signs) as the parasite likely is dead already
- Can use dexamethasone instead of prednisone



FOLLOW-UP CARE

PATIENT MONITORING

- Sequential nervous system evaluations

PREVENTIONS AND AVOIDANCE

- Keep cats indoors

POSSIBLE COMPLICATIONS

- May continue to have uncontrolled seizures
- May continue to circle compulsively
- May have behavioral changes, such as aggression

EXPECTED COURSE AND PROGNOSIS

- After initial onset of clinical signs, many patients improve and become acceptable pets; however, persistent nervous system deficits, seizures, circling and undesirable behavior (such as aggression) may continue
- Persistent clinical signs depend on the damage caused by the loss of blood flow to the brain (ischemia) and the actual parasitic migration through the nervous tissue



KEY POINTS

- Only occurs in outdoor cats and cats with access to the outdoors; indoor cats do not develop feline ischemic encephalopathy
- Only occurs in summer months with the majority of patients seen during July, August and September in the northeast United States and southeast Canada
- May not occur in major metropolitan areas that do not have the normal appropriate hosts (such as the cottontail rabbit) for *Cuterebra*

FELINE LEUKEMIA VIRUS (FeLV) INFECTION



BASICS

OVERVIEW

- A retrovirus that causes inability to develop a normal immune response (known as “immunodeficiency”) and development of tumors in domestic cats

GENETICS

- No genetic susceptibility to infection by feline leukemia virus (FeLV)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Breed Predilections

- None

Mean Age and Range

- Number of cases highest between 1 and 6 years of age
- Mean—3 years of age

Predominant Sex

- Male-to-female ratio—1.7:1 (that is, males are 1.7 times more likely to have feline leukemia virus [FeLV] infection than are females)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Onset of feline leukemia virus (FeLV)-associated disease—usually occurs over a period of months to years after infection
- Associated diseases—may be related to inability to develop a normal immune response (immunodeficiency) or to development of tumors or cancer
- Clinical signs of feline leukemia virus (FeLV)-induced inability to develop a normal immune response (immunodeficiency) cannot be distinguished from those of feline immunodeficiency virus (FIV)-induced immunodeficiency
- Signs depend on the type of disease (inability to develop a normal immune response [immunodeficiency] or tumor/cancer) and occurrence of secondary infections
- Enlarged lymph nodes (known as “lymphadenomegaly”)—mild to severe
- Upper respiratory tract disease—inflammation of the nose (known as “rhinitis”), inflammation of the moist tissues of the eye (known as “conjunctivitis”), and inflammation of the cornea (known as “keratitis;” the “cornea” is the clear outer layer of the front of the eye), seen in 18% of cases
- Persistent diarrhea
- Inflammation of the gums (known as “gingivitis”), of the mouth (known as “stomatitis”), and/or of the tissues surrounding and supporting the teeth (known as “periodontitis”)
- Long-term (chronic), nonresponsive or recurrent infections of the external ear and skin; abscesses
- Fever and wasting (seen in 42% to 53% of cases)
- Lymphoma (a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body)—most common feline leukemia virus (FeLV)-associated cancer
- Leukemia
- Fibrosarcomas (cancer that develops from fibrous tissue)—in patients co-infected with mutated sarcoma virus; most frequently in young cats
- Disorders usually affecting the nerves to the legs and paws (known as “peripheral neuropathies”); progressive wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)

CAUSES

- Cat-to-cat transmission—bites; close casual contact (such as grooming); shared dishes or litter pans
- Transmission of the virus from the mother cat (known as a “queen”) around the time of birth—fetal and newborn kitten death from 80% of affected queens; transmission across the placenta or through the milk in at least 20% of surviving kittens from infected queens

RISK FACTORS

- Age—kittens are much more susceptible to infection than are adults

- Male—result of behavior
- Cat allowed outside; free roaming cats
- Multicat household



TREATMENT

HEALTH CARE

- Outpatient for most cats
- Inpatient—may be required with severe secondary infections, low red-blood cell count (known as “anemia”), or extreme weight loss with muscle wasting (known as “cachexia”) until condition is stable
- Blood transfusions—emergency support; multiple transfusions may be necessary
- Management of secondary and opportunistic infections—primary consideration; “opportunistic infections” are infections caused by organisms that usually do not cause disease, but are able to cause disease because the animal’s body and/or immune system has been weakened, in this case by the feline leukemia virus (FeLV) infection
- Supportive therapy (such as fluids and nutritional supplements) may be useful

ACTIVITY

- Normal

DIET

- Normal
- Diarrhea, kidney disease, or long-term (chronic) wasting—may require special diet

SURGERY

- Biopsy or surgical removal of tumors
- Dental cleaning, tooth extraction, biopsy of the gums



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Zidovudine (Retrovir®)—antiviral agent; may lead to clinical improvement, but does not clear virus
- Medications to alter the immune response (known as “immunomodulatory drugs”)—may alleviate some clinical signs; interferon (Roferon®) may increase survival rates and improve clinical status; *Propionibacterium acnes* (ImmunoRegulin®); acemannan (Carrisyn™)
- *Mycoplasma felis* infection—suspect in all cats with low red-blood cell counts due to the destruction of red-blood cells, in which the body is producing new red-blood cells (known as “regenerative hemolytic anemias”); oxytetracycline or doxycycline; short-term use of steroids, administered by mouth, if needed
- Lymphoma (a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body)—management with standard combination chemotherapy protocols; periods of remission average 3 to 4 months; some cats may remain in remission for much longer
- Blood disorders or disease and leukemias—less responsive to medical treatment; for low red-blood cell count (anemia), try erythropoietin (Epogen®); for low neutrophil count (known as “neutropenia”), try Neupogen®
- Yearly vaccination for respiratory and intestinal viruses with inactivated vaccines recommended



FOLLOW-UP CARE

PATIENT MONITORING

- Varies according to the secondary infections and other manifestations of disease

PREVENTIONS AND AVOIDANCE

- Prevent contact with feline leukemia virus (FeLV)-positive cats
- Quarantine and test incoming cats before introduction into households currently with one or more cats

Vaccines

- Several commercial feline leukemia virus (FeLV) vaccines are available
- Test cats for FeLV before initial vaccination; if pre-vaccination testing is not done, advise clients that the cat may already be infected

POSSIBLE COMPLICATIONS

- Exposure of non-feline leukemia virus (FeLV)-infected cats to infection
- Development of disease related to inability to develop a normal immune response (immunodeficiency)
- Development of tumors or cancer
- Death

EXPECTED COURSE AND PROGNOSIS

- Cats that persistently have feline leukemia virus (FeLV) in their blood (known as “FeLV viremic cats”)—more than 50% succumb to related diseases within 2 to 3 years after infection



KEY POINTS

- Keep feline leukemia virus (FeLV)-infected cats indoors and separated from FeLV-negative cats, to protect them from exposure to secondary disease-causing agents and to prevent spread of FeLV to other cats
- Good nutrition is important
- Control secondary bacterial, viral, and parasitic infections

FELINE PANLEUKOPENIA



BASICS

OVERVIEW

- A sudden (acute), viral intestinal infection of cats, characterized by sudden onset, depression, vomiting and diarrhea, severe dehydration, and high mortality
- Usually caused by infection with feline parvovirus

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats—all, domestic and exotic
- Others—mink and some exotic dogs may be susceptible to feline panleukopenia infection; raccoons and coatimundi are susceptible to feline panleukopenia infection

Mean Age and Range

- Unvaccinated and previously unexposed cats of any age can become infected
- Kittens 2 to 6 months of age—most susceptible to develop severe disease
- Adults—often mild or subclinical infection; a “subclinical infection” is one for which animal is infected, but has no signs of disease

SIGNS/OBSERVED CHANGES in the ANIMAL

- Newly acquired kitten; potential of recent exposure to virus (such as at an adoption shelter or from a facility with history of feline panleukopenia)
- Sudden onset, with vomiting, diarrhea, depression, and complete lack of appetite (known as “anorexia”)
- Owner may suspect poisoning.
- Cat may have disappeared or hidden for 1 day or more before being found
- Cat hangs head over water bowl or food dish, but does not eat or drink
- Depression—may be mild to severe
- Typical “panleukopenia posture”—chest and chin resting on floor, feet tucked under body, and top of shoulders elevated above the back
- Dehydration—appears rapidly; may be severe
- Body temperature—usually mild to moderately elevated or depressed in the early stages of disease; becomes severely subnormal as affected cat reaches point of death (known as being “moribund”)
- Abdominal pain
- Small intestines—either turgid and hose-like or flaccid
- Subclinical or mild infections with few or no clinical signs common, especially in adults
- Wobbly, incoordinated or “drunken” appearing gait or movement with lack of coordination of the limbs, head, and/or trunk (known as “ataxia”) from underdevelopment of the cerebellum (the part of the brain responsible for body posture and balance; condition known as “cerebellar hypoplasia”)—kittens infected with feline parvovirus while in the uterus or as newborns; signs evident at 10 to 14 days of age and persist for life: overreaching or overstepping (known as “hypermetria”); incoordination with a base-wide stance and an elevated “rudder” tail; alert and otherwise normal; abnormal development of the back part of the eye (retina; condition known as “retinal dysplasia”) sometimes seen

CAUSES

- Feline parvovirus
- Canine parvovirus Types 2a, 2b, and 2c can produce feline panleukopenia in domestic and exotic cats

RISK FACTORS

- Intestinal parasites; intestinal disease-causing bacteria
- Secondary or coexistent infections—such as viral upper respiratory infections
- Age—kittens 2 to 6 months of age tend to be affected more severely



TREATMENT

HEALTH CARE

- Main principles of treatment—rehydration; reestablishment of electrolyte balance; supportive care until the patient's immune system produces antiviral antibodies that neutralize the virus
- Inpatient—severe cases; hydration and replacement electrolyte therapy
- Outpatient—mild cases
- Fluid therapy—essential in severe cases; with electrolyte replacement and intravenous nutrient support may well make the difference between survival and death
- Whole blood transfusions—may be necessary if plasma protein levels drop too low or if total white-blood cell (WBC) count is too low (less than 2,000 cells/dl)

ACTIVITY

- Keep patient indoors during the sudden (acute) disease—prevent contamination of the environment; prevent the cat from going into hiding

DIET

- Temporarily withhold food until the sudden (acute) inflammation of the stomach and intestines (known as “gastroenteritis”) is controlled



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Broad-spectrum antibiotics—counter the presence of bacteria in the blood (known as “bacteremia”) that enter the bloodstream from the diseased intestines



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor hydration and electrolyte balance closely
- Monitor complete blood count (CBC) daily or at least every 2 days until recovery

PREVENTIONS AND AVOIDANCE

- Contaminated environments (such as cat carriers, cages, floors, food and water dishes) should be disinfected with a 1:32 dilution of household bleach
- Parvovirus is resistant to most commercial disinfectants

Vaccines

- Completely preventable by routine vaccination of kittens
- Modified live virus (MLV) or inactivated virus vaccines are available; vaccines may be administered by injection or into the nose (intranasal vaccine), as directed by the manufacturer
- Immunity—long duration, perhaps even for life
- Kittens—vaccinate at 8 to 10 weeks of age; then after 12 weeks of age
- Boosters—after 1 year; repeat every 3 years to provide excellent immunity, as directed by your cat's veterinarian

POSSIBLE COMPLICATIONS

- Long-term (chronic) inflammation of the intestines (known as “enteritis”)—fungal or other cause
- Abnormal development of the fetus caused by the virus (known as “teratogenic effects”), such as underdevelopment of the cerebellum

(cerebellar hypoplasia) resulting in a wobbly, “drunken” appearing gait (ataxia) for life—virus infection of fetus; also may see signs with infection of the newborn kitten

- Shock and other complications—severe dehydration and electrolyte imbalance

EXPECTED COURSE AND PROGNOSIS

- Most cases are sudden (acute), lasting only 5 to 7 days
- If death does not occur during the sudden (acute) disease, recovery is usually rapid and uncomplicated; it may take several weeks for the patient to regain weight and body condition
- Prognosis is guarded during the sudden (acute) disease, especially if the total white-blood cell (WBC) count is less than 2,000 cells/dl
- Recovered cats are immune against feline parvovirus (panleukopenia) infection for life



KEY POINTS

- All current and future cats in the household must be vaccinated against feline parvovirus (panleukopenia) before exposure
- Feline parvovirus is extremely stable against environmental factors, temperature, and most disinfectants
- The virus will remain infectious on the premise for years, unless the environment can be disinfected adequately with household bleach

FELINE RHINOTRACHEITIS—FELINE HERPESVIRUS-1 INFECTION



BASICS

OVERVIEW

- “Rhino-” refers to the nose; “tracheitis” is inflammation of the windpipe or trachea; “rhinotracheitis” is inflammation of the nose and windpipe
- Sudden (acute) disease in domestic and exotic cats, which is characterized by sneezing, fever, inflammation of the nose (known as “rhinitis”), inflammation of the moist tissues of the eyes (known as “conjunctivitis”), and changes in the cornea (the clear outer layer of the front of the eye) characterized by the presence of ulcers, with or without inflammation (condition known as “ulcerative keratitis”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Affects all domestic and many exotic cats

Breed Predilections

- None

Mean Age and Range

- Cats of all ages
- Kittens most susceptible to infection

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden (acute) onset of paroxysmal sneezing (that is, sneezing episodes occur suddenly at fairly regular intervals)
- Squinting or spasmodic blinking (known as “blepharospasm”) and discharge from the eyes
- Lack of appetite (known as “anorexia”)—from high fever, general signs of discomfort and “not feeling well” (known as “malaise”), or inability to smell
- Recurrent signs—carriers (cats that have no signs of rhinotracheitis, but harbor the feline herpesvirus and can transmit it to other cats)
- Abortion
- Fever—up to 106° F (41° C)
- Inflammation of the nose (rhinitis)—discharge from the nose; it may be clear or may contain mucus and/or pus
- Inflammation of the eyes (conjunctivitis)—discharge from the eyes; it may be clear or may contain mucus and/or pus
- Long-term (chronic) inflammation of the nose (rhinitis) and of the sinuses (known as “sinusitis”)—long-term (chronic) discharge from the nose that contains pus
- Inflammation of the cornea, the clear outer layer of the front of the eye (known as “keratitis”)—ulceration of the cornea; extremely deep ulceration of the cornea, to the inner most membrane of the cornea (known as a “descemetocoele”), which may lead to rupture of the eye; or inflammation of all parts of the eye (known as “panophthalmitis”)

CAUSES

- Feline herpesvirus-1 (FHV-1)

RISK FACTORS

- Lack of vaccination for feline herpesvirus-1
- Facilities with multiple cats with overcrowding, poor air exchange (ventilation), poor sanitation, poor nutrition, or physical or psychological stress
- Pregnancy and nursing (lactation)
- Coexistent disease, especially owing to disease-causing agents that decrease the ability to develop a normal immune response (known as being “immunosuppressive”) or cause other respiratory disease
- Kittens born to mother cats (known as “queens”) that have no signs of rhinotracheitis, but harbor the feline herpesvirus and can transmit it (known as “carrier queens”)—infected about 5 weeks of age



TREATMENT

HEALTH CARE

- Inpatient—nutritional and fluid support to cats with lack of appetite (anorexia); prevent spread of the virus (known as “contagion”)
- Outpatient—keep patient indoors to prevent environmentally induced stress, which may lengthen the course of the disease
- Fluids—intravenous or subcutaneous (that is, administered under the skin); to correct or prevent dehydration; to keep nasal secretions thin

ACTIVITY

- Isolate affected cats, because they can spread the virus to other cats (that is, they are contagious)

DIET

- Outpatient—entice food consumption to avoid lack of appetite (anorexia), which induces a cascade of negative consequences; offer foods with appealing tastes and smells
- Inpatients—forced feeding for cats that have lack of appetite (anorexia); carefully clean discharge from the nose (so nasal breathing can occur) before starting tube feeding

SURGERY

- Surgically implanted feeding tubes may be needed when prolonged lack of appetite (anorexia) occurs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Broad-spectrum antibiotics—amoxicillin for secondary bacterial infections
- Antibiotic combinations—amoxicillin and enrofloxacin for secondary bacterial infections
- Lysine may have some effect against the virus; “lysine” is an amino acid (the smallest component of protein); treatment with lysine may decrease eye problems related to feline rhinotracheitis and may decrease the activity of the virus
- Antibiotics applied directly to the eye (topical treatment)—for inflammation of the cornea (keratitis)
- Medications to eliminate the virus applied directly to the eye (known as “ophthalmic antivirals”)—idoxuridine, trifluridine; for ulcers caused by herpesvirus; must be administered every 2 hours for significant effect
- Some evidence suggests that administration of an intranasal vaccine 2 to 6 days prior to exposure will result in lessening of clinical signs; such use may be helpful in an outbreak in a multicat setting
- α -Interferon—Roferon®; some effectiveness in controlling the viral aspect of long-term (chronic) infectious nasal discharge



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor appetite closely; hospitalize for feeding if lack of appetite (anorexia) develops

PREVENTIONS AND AVOIDANCE

- Routine vaccination with a modified live virus (MLV) vaccine or inactivated virus vaccine—prevents development of severe disease; does not prevent infection and local viral replication with mild clinical disease and virus shedding
- Vaccinate at 8 to 10 weeks of age; at 12 to 14 weeks of age; and with boosters, as directed by your cat’s veterinarian
- In facilities or households with multiple cats that have cats with feline rhinotracheitis or history of infection—vaccinate kittens with a dose of an intranasal vaccine at 10 to 14 days of age; then vaccinate using an injectable vaccine at 6, 10, and 14 weeks of age; isolate the litter from *all* other cats at 3 to 5 weeks of age; then use kitten vaccination protocol to prevent early infections

POSSIBLE COMPLICATIONS

- Long-term (chronic) inflammation of the nose and sinuses (known as “rhinosinusitis”) with long-term sneezing and nasal discharge
- Changes in the cornea (the clear outer layer of the front of the eye) caused by herpesvirus infection, characterized by the presence of ulcers, with or without inflammation (condition known as “herpetic ulcerative keratitis”)
- Permanent closure of the outflow portion of the drainage system that normally moves tears to the nasal passages (known as the “nasolacrimal duct”) with long-term (chronic) eye discharge

EXPECTED COURSE AND PROGNOSIS

- Usually 7 to 10 days before spontaneous remission, if secondary bacterial infections do not occur
- Prognosis generally good, if fluid and nutritional therapy are adequate



KEY POINTS

- Feline rhinotracheitis is a contagious disease
- Proper vaccination protocols and early vaccination to cats in multicat facilities and households are necessary to decrease severity of disease; vaccination does not prevent infection by feline herpesvirus-1
- Early weaning and isolation from all other cats except litter mates may prevent infections

FLATULENCE (EXCESSIVE GAS IN THE DIGESTIVE TRACT)



BASICS

OVERVIEW

- Excessive gas formation in the stomach or intestines (known as “flatulence”)
- Burping or belching (known as “eructation”) is the passage of gas from the stomach through the mouth
- Expelling or passing gas (known as “flatus”) through the anus

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Common complaint in dogs; rare in cats

Breed Predisposition

- Excessive swallowing of air (known as “aerophagia”) is seen in short-nosed, flat-faced (brachycephalic) breeds, sporting dogs, and those with excessive eating or drinking behavior (known as “gluttonous behavior”) and with competitive-eating behaviors

Mean Age and Range

- Any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Increased frequency and possibly volume of gas expelled or passed through the anus (flatus) as detected by the pet owner
- Mild abdominal discomfort caused by gastrointestinal distention possible
- When increased frequency and possibly volume of gas expelled or passed through the anus (flatus) is due to gastrointestinal disease, may see additional gastrointestinal signs—diarrhea, vomiting, rumbling or gurgling sounds in the intestines (known as “borborygmus”), and weight loss

CAUSES

Excessive Swallowing of Air (Aerophagia)

- Excessive eating or drinking (gluttony) or competitive eating
- Respiratory disease or any cause of increased breathing rate
- Feeding shortly after exercise
- Short-nosed, flat-faced (brachycephalic) breeds

Diet-Related

- Diets high in partially digestible vegetable sugars (nonabsorbable oligosaccharides)—soybeans, peas, beans
- Diets high in fermentable fiber—lactose, Fibrim®, psyllium, oat bran
- Spoiled diets
- Milk products
- Abrupt changes in diet
- Spices and food additives/supplements

Disease Conditions

- Sudden (acute) and long-term (chronic) intestinal disease—inflammatory bowel disease (IBD); increased number of bacteria in the small intestine (known as “small intestinal bacterial overgrowth”); cancer; irritable-bowel syndrome; parasitism; bacteria-caused inflammation of the intestines (known as “bacterial enteritis”); and virus-caused inflammation of the intestines (known as “viral enteritis”)
- Inadequate production of digestive enzymes by the pancreas (known as “exocrine pancreatic insufficiency”)

RISK FACTORS

- Nervous, excessive eating or drinking behavior (gluttonous behavior), or competitive eating
- Eating soon after exercise
- Short-nosed, flat-faced (brachycephalic) breeds
- Abrupt changes in diet
- Inappropriate or spoiled foods
- Sedentary lifestyle—a 1998 study (Jones, et al) reported that 43% of randomly chosen dog owners detected the expelling or passing of gas through the anus (flatus) most commonly in sedentary pets, and with no association to a particular diet



TREATMENT

HEALTH CARE

- Outpatient
- Treat any underlying gastrointestinal disease

ACTIVITY

- Encourage an active lifestyle—exercise increases gastrointestinal motility, which will help expel intestinal gas and increase regularity of bowel movements

DIET

- Feed small, more frequent meals in an isolated, quiet environment
- Change diet to a highly digestible, low-fiber and low-fat diet (such as Eukanuba® Low Residue Formula™, Hill's Prescription Diet® i/d®, Purina Veterinary Diets® EN Gastro ENteric® Canine Formula, Royal Canin Veterinary Diet™ Low Fat formula), or feed homemade diets containing boiled white rice (dogs) or baby cereal (cats) with skinned chicken or cottage cheese (balanced with vitamins and minerals).
- Change in protein or carbohydrate source of diet benefits some individuals
- In cats, high-protein, low-carbohydrate diets may be beneficial



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Carminitives are medications that relieve excessive gas in the stomach and intestines (flatulence)
- Zinc acetate binds sulfur-containing compounds
- *Yucca schidigera* binds ammonia and is added to pet foods as a flavoring agent
- Inclusion of activated charcoal, *Yucca schidigera*, and zinc acetate in a treat reduced the frequency of highly odiferous episodes in dogs
- Bismuth subsalicylate adsorbs hydrogen sulfide and has antibacterial properties; however, the required long-term, multiple daily dosing makes it impractical
- Simethicone is an antifoaming agent that reduces surface tension of gas bubbles, allowing easier coalescence and release of intestinal gas; however, gas production is unaltered
- Pancreatic digestive enzyme supplements may reduce gas in the stomach and intestines (flatulence) in some patients
- More than 30 herbal and botanical preparations are available to reduce gas in the stomach and intestines (flatulence); however, the dosage, safety, and efficacy are unknown



FOLLOW-UP CARE

PATIENT MONITORING

- Response to therapy

PREVENTIONS AND AVOIDANCE

- Avoid diets high in partially digestible vegetable sugars (nonabsorbable oligosaccharides) and high in fermentable fiber
- Avoid milk products, spoiled diets, and abrupt changes in diet
- Do not feed shortly after exercise

POSSIBLE COMPLICATIONS

- None



KEY POINTS

- Excessive gas formation in the stomach or intestines
- Burping or belching (eructation) is the passage of gas from the stomach through the mouth
- Expelling or passing gas (flatus) through the anus
- Discourage dietary indiscretions (such as garbage ingestion or eating feces [known as “coprophagia”])
- Avoid diets high in partially digestible vegetable sugars (nonabsorbable oligosaccharides) and high in fermentable fiber
- Avoid milk products, spoiled diets, and abrupt changes in diet
- Do not feed shortly after exercise

FLEA-BITE HYPERSENSITIVITY AND FLEA CONTROL



BASICS

OVERVIEW

- “Hypersensitivity” is an increased sensitivity or reaction in the skin due to the presence of a foreign substance; in flea-bite hypersensitivity, the foreign substance is found in flea saliva; the reaction is immune based and would be considered to be an “allergic” reaction
- “Dermatitis” is the medical term for inflammation of the skin
- “Antigens” are substances that induce sensitivity or immune response
- “Flea-bite hypersensitivity” or “flea-allergy dermatitis”—hypersensitivity reaction to antigens in flea saliva, with or without evidence of fleas and flea dirt
- “Flea infestation”—fleas and flea dirt are present on the pet, with or without signs of flea-allergy dermatitis
- “Flea-bite dermatitis”—inflammation of the skin due to the flea bite itself; it is not an allergic or hypersensitivity reaction, but rather an irritant response to flea bites

GENETICS

- Flea-bite hypersensitivity—unknown inheritance pattern; more common in breeds with atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Flea-bite hypersensitivity—rare in pets less than 6 months of age; average age range, 3 to 6 years, but may be seen at any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend somewhat on the severity of the reaction and the degree of exposure to fleas (that is, seasonal or year-round)
- Itchiness (known as “pruritus”)
- Compulsive biting
- Chewing (“corn cob nibbling”)
- Licking, primarily in the back half of the body, but may include other areas
- Cats—scratching around the head and neck
- Signs of fleas and flea dirt; finding fleas and flea dirt is beneficial, although not essential, for the diagnosis of flea-bite hypersensitivity; sensitive animals require a low exposure to fleas to have an immune response and they tend to over groom, removing evidence of flea infestation, and making identification of parasites difficult
- Hair loss (known as “alopecia”)
- Small, raised skin lesions (known as “papules”)
- Darkened skin (known as “hyperpigmentation”) in dogs
- Thickening and hardening of the skin, usually associated with hyperpigmentation (known as “lichenification”) in dogs
- “Hot spots” in dogs
- Miliary dermatitis (skin inflammation characterized by numerous, small, crusty bumps) in cats

CAUSES

- Fleas
- Immune response to flea saliva (flea-bite hypersensitivity or flea-allergy dermatitis)

RISK FACTORS

- Flea-bite hypersensitivity—intermittent exposure to fleas increases likelihood of development; commonly seen in conjunction with atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems)



TREATMENT

HEALTH CARE

- Outpatient treatment



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids—to decrease inflammation in the skin for symptomatic relief, while fleas are being controlled
- Antihistamines—symptomatic relief
- Flea control on the pet; examples include fipronil (monthly spot treatment for dogs and cats and spray treatment for dogs); imidacloprid (monthly spot treatment for cats and dogs); lufenuron (oral formulation for dogs and cats and as an injection for cats); selamectin (monthly spot treatment for dogs and cats)
- Other flea control for pets (such as flea sprays)—usually contain pyrethrins and pyrethroids (synthetic pyrethrins) with an insect-growth regulator or synergist; generally effective for less than 48 to 72 hours
- Environmental/indoor treatment—professional exterminator or home-use fogs and premises sprays; usually contain organophosphates, pyrethrins, and/or insect-growth regulators; apply according to manufacturer's directions; treat all areas of the house
- Environmental/indoor treatment using inert substances—boric acid, diatomaceous earth, and silica aerogel; treat every 6 to 12 months; follow manufacturer's recommendations
- Environmental/outdoor treatment—concentrate outdoor treatment in shaded areas; sprays usually contain pyrethroids or organophosphates and an insect-growth regulator; a product containing nematodes (*Steinernema carpocapsae*) is chemical-free
- Other products for use include flea powders, dips, sprays, and foams
- **NOTE:** Always read all label instructions and follow the manufacturer's directions when using any flea-control product; ensure that the product is labeled for use on the species (dog or cat) or location (indoors or outdoors) for which you intend to apply it—for example, some products that are safe for dogs are very toxic to cats and should not be used on cats



FOLLOW-UP CARE

PATIENT MONITORING

- Itchiness (pruritus)—a decrease in itchiness indicates the flea infestation and/or flea-bite hypersensitivity is being controlled
- Fleas and flea dirt—absence is not always a reliable indicator of successful treatment in very sensitive animals

PREVENTIONS AND AVOIDANCE

- Year-round warm climates—year-round flea control
- Seasonally warm climates—begin flea control in May or June, as directed by your pet's veterinarian

POSSIBLE COMPLICATIONS

- Secondary bacterial infections
- Sudden (acute) moist dermatitis, also known as “hot spots”
- Acral lick dermatitis (inflammation of the skin characterized by a firm, ulcerated lesion on a leg, caused by constant licking)

EXPECTED COURSE AND PROGNOSIS

- Prognosis is good, if strict flea control is instituted



KEY POINTS

- Flea control is important for dogs and cats
- No cure exists for flea-allergy dermatitis or flea-bite hypersensitivity
- Flea-allergic animals often become more sensitive to flea bites as they age
- Controlling exposure to fleas is currently the only means of controlling signs; “allergy shots” (known as “hyposensitization”) for flea-allergy dermatitis or flea-bite hypersensitivity have not worked satisfactorily

GASTROINTESTINAL FOOD REACTIONS

(REACTIONS TO FOODS THAT LEAD TO STOMACH OR INTESTINAL PROBLEMS)



BASICS

OVERVIEW

The body can react negatively to substances, such as particular foods or ingredients of a food. These negative reactions can be due to true allergic reactions, in which the immune system develops a response to the food (known as “dietary allergy”). By definition, dietary allergy is an “immunologic” reaction, because the immune system responds to the particular offending substance in the food. Other negative reactions are not true allergic reactions. The body still reacts to the foods or ingredients of a food, but the immune system is not involved with the abnormal response. When the immune system is not involved, the negative responses are known as “nonimmunologic reactions.” These nonimmunologic reactions are called “dietary intolerance.” “Gastrointestinal food reactions” refers to the nonimmunologic reactions, and is characterized by the following:

- Abnormal reaction to food, not related to an immune response (that is a “nonimmunologic” reaction)
- Syndrome in which adverse clinical signs are associated with the inability to digest, absorb, and/or utilize a foodstuff or with an untoward reaction to a diet or component of a diet
- Dietary allergies or allergic reactions (that is, immunologic reactions) to food are differentiated from dietary intolerance (that is, nonimmunologic reactions) by the prominent immune component in the allergies
- In a practical sense, dietary allergy and dietary intolerance may have similar signs, causes, diagnostics, and treatments and may not be easily distinguishable

GENETICS

- Specifics of a genetic basis are not well defined
- Gluten-sensitive enteropathy (specific type of intestinal disease related to the presence of wheat gluten in the diet) has been seen primarily in Irish setters
- Siamese and Siamese-cross cats may be at increased risk, as compared to other breeds of cats, to develop gastrointestinal food reactions

SIGNALMENT/DESCRIPTION of ANIMAL

- Cats and dogs of any age, breed or sex
- More common in cats than in dogs
- Irish setters are predisposed to gluten-sensitive enteropathy; they tend to display clinical signs by 4 to 7 months of age
- Juvenile cats and dogs have higher lactase (the enzyme that breaks down milk sugar [lactose] during digestion) activity and are less likely to display lactose intolerance, which is common in adults of both species

SIGNS/OBSERVED CHANGES in the ANIMAL

- Dietary intolerance commonly produces diarrhea (small or large bowel); vomiting; excessive gas formation in the stomach or intestines (flatulence); decreased appetite (anorexia); and abdominal discomfort
- Skin (dermatologic) changes, such as itchiness (known as “pruritus”); poor weight gain; and failure to thrive may be seen with long-term (chronic) dietary intolerance
- Sudden (acute) dietary intolerance may accompany feeding a foodstuff to which the animal has never been exposed (known as a “novel foodstuff;” for example, feeding a food that contains “alligator” as an ingredient and the animal has never eaten anything containing alligator), a new food source, or dietary change
- Clinical signs may cease when the animal is fasted or within days of a dietary change
- Physical examination findings are generally nonspecific; animal may have abdominal discomfort, excessive gas formation in the stomach or intestines (flatulence), gaseous bloating, or a poor body condition and evidence of weight loss
- Skin (dermatologic) responses in cats may include military dermatitis (frequently itchy condition, in which multiple bumps and/or crusts are present on the skin) and enlarged lymph nodes (known as “peripheral lymphadenopathy”)

CAUSES

- Reactions to food additives, where the individual pet is more likely to develop ill effects (known as “idiosyncratic reactions”)—for example, colorings, preservatives (such as butylated hydroxyanisole [BHA], monosodium glutamate, sodium nitrate, sulfur dioxide); spices; propylene glycol
- Drug reactions—substances that have an effect on the blood vessels (known as “vasoactive substances,” such as histamine); agents that have an effect on the nervous system to alter mood, anxiety or behavior (known as “psychoactive agents”); stimulants (such as theobromine, caffeine); other drugs

- Metabolic defects or deficiencies—enzyme defects in the intestinal tract (for example, lactase deficiency leading to lactose intolerance)
- Toxic reactions to foods or spoiled foods—spices; oxalate toxicity; lectin toxicity; aflatoxicosis (disease caused by eating foods contaminated with aflatoxin, poison produced by certain fungi, including *Aspergillus*); ergotism (disease caused by eating ergot-contaminated grains or grain products; ergot is a fungus that infects cereal plants); botulism (food poisoning caused by eating food containing botulin, a nerve toxin produced by a particular bacteria); dietary indiscretion

RISK FACTORS

- Young Irish setters susceptible to gluten-sensitive enteropathy (specific type of intestinal disease related to the presence of wheat gluten in the diet) may be at greater risk to develop the disease if exposed to gluten at an early age



TREATMENT

HEALTH CARE

- Patients with extreme and sudden (acute) signs may need hospitalization for intravenous fluid therapy, antibiotics, and supportive care
- Generally can treat on an outpatient basis

ACTIVITY

- No restrictions

DIET

- Feed a diet free of the offending ingredient(s)
- Cats generally are sensitive to more than one ingredient
- If no specific ingredient has been identified, feed a nutritionally complete exclusion diet (a diet that eliminates or excludes the food ingredient(s) to which the animal is intolerant)
- Can use trial-and-error to find a commercial diet that does not cause dietary intolerance
- If this approach is used, examination of the ingredients of the various diets being fed is recommended to determine if any patterns exist that might help identify the offending ingredient(s)



MEDICATIONS

- Generally no medications are used
- Associated problems (such as increased number of bacteria in the intestinal tract [known as “bacterial overgrowth”] or inflammatory bowel disease [IBD]) may require medical therapy



FOLLOW-UP CARE

PATIENT MONITORING

- Assess efficacy of treatment by observing improvement in clinical signs
- Consider repeating evaluation for primary disease (such as inflammatory bowel disease [IBD]) following dietary therapy

PREVENTIONS AND AVOIDANCE

- Avoid the offending food ingredient(s)
- If no specific ingredient has been identified, adhere to a particular exclusion diet

POSSIBLE COMPLICATIONS

- Antibiotic-responsive diarrhea (ARD) and inflammatory bowel disease (IBD)

EXPECTED COURSE AND PROGNOSIS

- Prognosis for a full recovery is excellent in most cases, if diet is found that the patient tolerates
- Rarely, severe reactions require short-term, aggressive in-hospital therapy



KEY POINTS

- Feed a diet free of the offending ingredient(s)
- Caution against feeding any scraps or varying from a set diet
- Prognosis for a full recovery is excellent in most cases, if diet is found that the patient tolerates

SKIN DISORDERS CAUSED BY REACTIONS TO FOOD



BASICS

OVERVIEW

- Nonseasonal skin disorders characterized by itchiness (known as “pruritus”), associated with ingestion of one or more substances in the animal’s food

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- A wide range of signs that can mimic any of the other increased sensitivities or reactions in the skin due to the presence of a foreign substance (known as “hypersensitivity”)
- Nonseasonal itchiness (pruritus) of any body location
- Poor response to anti-inflammatory doses of steroids in the treatment of suspected “allergy,” suggests a food hypersensitivity
- Vomiting
- Diarrhea
- Excessive rumbling or gurgling sounds caused by movement of gas in the intestinal tract (known as “borborygmus”), excessive gas formation in the stomach or intestines (known as “flatulence”), and frequent bowel movements
- Inflammation of the skin (known as “dermatitis”) with the presence of the yeast, *Malassezia*
- Skin infection characterized by the presence of pus (known as “pyoderma”)
- Inflammation of the outer ear (known as “otitis externa”)
- Thickened, raised, flat-topped areas that are slightly higher than normal skin (known as “plaques”)
- Small, circumscribed elevations of the outer layer of the skin (epidermis) filled with pus (known as “pustules”)
- Reddening of the skin (known as “erythema”)
- Dried discharge on the surface of skin lesions (known as “crusts”)
- Accumulations of surface skin cells, such as seen in dandruff (known as “scales”)
- Self-induced hair loss (known as “self-induced alopecia”)
- Scratches or breaks in the skin (from scratching and/or biting at the skin), frequently with blood or dried discharge (known as “excoriations”)
- Darkened skin (known as “hyperpigmentation”)
- Thickening and hardening of the skin, usually associated with hyperpigmentation (known as “lichenification”)
- Hives or wheals (known as “urticaria”)
- Large, localized areas of fluid build-up under the skin (known as “angioedema”)

CAUSES

- Immune-mediated reactions—result of the ingestion and subsequent presentation of one or more proteins (allergens) either before or after digestion; sensitization may occur at the lining of the gastrointestinal tract, after the substance is absorbed, or both; “allergens” are substances to which the animal has developed an allergy
- Nonimmune (food intolerance) reactions—result of ingestion of foods with high levels of histamine or substances that induce histamine either directly or through histamine-releasing factors; the pet reacts to the food, but the reaction is not a true allergic reaction

RISK FACTORS

- Unknown
- Speculated that intestinal parasites or intestinal infections in juvenile animals may cause damage to the lining of the intestines, resulting in abnormal absorption of allergens and subsequent sensitization of the animal to a particular substance and leading to a “food allergy” to that substance



TREATMENT

HEALTH CARE

- Outpatient management
- Other causes of itchiness (pruritus), such as flea-bite allergy; atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems); and external parasites also must be treated to control signs

ACTIVITY

- No change

DIET

- Avoid any food substances that cause clinical signs to return
- Dietary trial or food elimination diet trial may be necessary to identify proteins/allergens to which the animal reacts
- “Elimination diet” is a diet that does not contain substances that the animal normally eats and is free of additives
- “Novel protein diet” uses a novel protein source (that is, feeding a protein to which the animal has never been exposed) or hydrolyzed protein diet (where the protein source has been processed to break down the protein into smaller units, less likely to cause an allergic response) as a dietary trial or as part of the animal’s treatment



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drugs administered by mouth or injection to control the itchiness (known as “systemic antipruritic drugs”)—may be useful during the first 2 to 3 weeks of diet trial to control self-mutilation
- Antibiotics or antifungal medications—useful for secondary skin infections characterized by the presence of pus (pyodermas) or *Malassezia* (yeast) infections



FOLLOW-UP CARE

PATIENT MONITORING

- Examine patient and evaluate levels of itchiness (pruritus) and clinical signs every 3 to 4 weeks

PREVENTIONS AND AVOIDANCE

- Avoid intake of any of the proteins included in the previous diet
- Treats and chewable toys should be limited to known safe substances (such as apples and/or vegetables used as treats)
- Chewable vitamins and heartworm medications may contain offending food substances and may need to be discontinued; follow the recommendations of your pet’s veterinarian

POSSIBLE COMPLICATIONS

- Other causes of itchiness (pruritus), such as flea-bite allergy; atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems); and external parasites (such as sarcoptic, *Notoedres*, and *Cheyletiella* mites) can mask the response to the food elimination diet trial
- Rarely a dog or cat may develop increased sensitivity or reaction (hypersensitivity) to new substances, which may require a new elimination diet trial

EXPECTED COURSE AND PROGNOSIS

- Prognosis is good, if food ingredients are the only cause of the itchiness (pruritus) and the offending ingredients are avoided



KEY POINTS

- Eliminate treats, chewable toys, vitamins, and other chewable medications (such as heartworm preventive), which may contain ingredients from the patient's previous diet; elimination of chewable medications should be done only under the direction of your pet's veterinarian
- Outdoor pets must be confined to prevent foraging and hunting, which might alter the test diet during the diet trial and might alter the maintenance diet
- All family members must be aware of the diet trial test protocol and must help keep the test diet clean and free of any other food sources

ESOPHAGEAL FOREIGN BODIES



BASICS

OVERVIEW

- Eating (ingestion) of foreign material or foodstuffs too large to pass through the esophagus (the tube running from the throat to the stomach), causing blockage within the open space of the tube (known as “intraluminal obstruction”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs are more likely to have esophageal foreign bodies than are cats due to their indiscriminate eating habits

Breed Predilection

- More common in small-breed dogs; terrier breeds tend to be more likely to have esophageal foreign bodies than other breeds

Mean Age and Range

- More common in young to middle-aged animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Observation of pet eating or ingesting a foreign body
- Unsuccessful attempts to vomit (known as “retching”); gagging; sluggishness (lethargy); lack of appetite (anorexia); drooling (known as “ptyalism”); regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth); restlessness; difficulty swallowing (known as “dysphagia”); and persistent gulping
- Occasionally discomfort will be noted when feeling (palpating) the neck or cranial abdomen

CAUSES

- Occurs most often with an object for which size, shape, or texture does not allow free movement through the esophagus, causing the object to become lodged before it can pass into the stomach



TREATMENT

HEALTH CARE

- Emergency care—treat as inpatients and perform an examination of the open space of the esophagus using a special instrument called an “endoscope” (general term for procedure is “endoscopy”) as soon as possible after diagnosis
- If retrieval of the foreign body using the endoscope succeeds *and* esophageal damage is minimal, the patient may be discharged the same day with no special aftercare needed

ACTIVITY

- The patient routinely may resume normal activity after a foreign body has been removed

DIET

- No change needed in most cases
- Severe trauma to the lining of the esophagus (mucosal trauma) may require using a feeding tube to allow nutritional support during esophageal healing

SURGERY

- Endoscopy is much less traumatic and invasive than surgery
- Surgery is indicated when endoscopy fails to retrieve the foreign body; when endoscopy enables advancement of the object into the stomach, but the foreign body is too large to pass through the gastrointestinal tract; or when a large tear in the esophagus (known as “esophageal perforation”) or area of dead tissue (known as “necrosis”) requires surgical repair
- It often is less traumatic to advance a bone foreign body into the stomach than to attempt retrieval; many bone foreign bodies safely can be left to dissolve in the stomach without need for surgical removal



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

In cases with significant injury to the lining of the esophagus and/or ulceration of the lining of the esophagus, recommendations include the following:

- Broad-spectrum antibiotics, such as amoxicillin or Clavamox®
- Sucralfate slurry to protect the lining of the esophagus and to allow healing
- Short-term corticosteroids decrease the risk of stricture formation by inhibiting fibroblasts; contraindicated if animal has aspiration pneumonia
- H₂-blockers or antagonists (such as ranitidine) for inflammation of the esophagus due to backward or reverse flow of stomach contents into the esophagus (known as “reflux esophagitis”)
- Metoclopramide for reflux esophagitis



FOLLOW-UP CARE

PATIENT MONITORING

- Examine the esophagus closely for damage to the lining
- Mild redness (known as “erythema”) and shallow ulcers (known as “erosions”) are not uncommon following esophageal foreign body, and tend to heal uneventfully
- Survey chest X-rays to assess for the presence of free air in the mediastinum, the area along the midline of the chest containing the heart and other structures of the chest, other than the lungs, (free air in the mediastinum is known as “pneumomediastinum”) or in the space between the chest wall and lungs (known as “pneumothorax”)
- Monitor at least 2 to 3 weeks for evidence of narrowing or scarring of the esophagus (stricture formation)
- Esophageal stricture—most common clinical sign is regurgitation; contrast X-ray studies of the esophagus (known as an “esophagram”) and/or follow-up endoscopic evaluation of the esophagus (known as “esophagoscopy”) may be indicated

PREVENTIONS AND AVOIDANCE

- Carefully monitor what is available in the environment (such as rocks or bones) that the pet might eat and take steps to prevent the pet from eating it
- Carefully monitor what is fed to the pet

POSSIBLE COMPLICATIONS

- Approximately 25% of patients with foreign bodies develop complications
- Complications most frequently encountered include tearing of the esophagus (esophageal perforation); narrowing or scarring of the esophagus (esophageal strictures); open tracts between the esophagus and the chest (known as “esophageal fistulas”); and severe inflammation of the esophagus (esophagitis)
- Localized, transient problems with normal movement of the esophagus can occur secondary to esophageal trauma
- Free air in the mediastinum (pneumomediastinum), free air in the space between the chest wall and lungs (pneumothorax), pneumonia, inflammation of the lining of the chest (known as “pleuritis”), inflammation of the mediastinum (known as “mediastinitis”), and tracts between the bronchus and the esophagus (known as “bronchoesophageal fistulas”) can occur secondarily to tearing or perforation of the esophagus

EXPECTED COURSE AND PROGNOSIS

- Most of these patients do well and recover uneventfully
- With complications, prognosis is guarded



KEY POINTS

- Dogs are more likely to have esophageal foreign bodies than are cats due to their indiscriminate eating habits
- Possibility of complications in approximately 25% of patients
- Possibility of another esophageal foreign body (that is, the animal is a “repeat offender”)

MAXILLARY AND MANDIBULAR FRACTURES

(UPPER AND LOWER JAW FRACTURES)



BASICS

OVERVIEW

- Fractures of the upper jaw (known as the “maxilla”), the lower jaw (known as the “mandible”), and associated structures are classified as to location, severity (that is, tooth involvement, soft-tissue tears, and type of bone fracture), and effects of the chewing muscles (known as “muscles of mastication”) on restoring the bones and teeth to their normal anatomic positions (known as “reduction”)
- “Occlusion” is the relationship or contact between the biting (known as “incising”) and chewing (known as “masticatory”) surfaces of the upper and lower teeth; “malocclusion” is any deviation in the relationship or contact between the biting and chewing surfaces of the upper and lower teeth

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary greatly according to the location, type, extent, cause of the fracture and underlying risk factors resulting in the injury
- Facial deformity; deviation in the relationship or contact between the biting and chewing surfaces of the upper and lower teeth (malocclusion); fractured teeth or teeth in abnormal positions; bleeding from the mouth or nose; and inability to properly close the mouth may be seen

CAUSES

- Injury, trauma, and predisposing factors, such as infections of the mouth and cancer

RISK FACTORS

- High-risk environment or temperament
- Oral infections (such as infection of the gums and supporting tissues of the teeth [known as “periodontal disease”] or infection/inflammation of the bone [known as “osteomyelitis”]); tumors or cancer; and certain metabolic diseases—may result in weaker jaws that are more prone to injury
- Traumatic injury affecting the jaws or teeth
- Congenital (present at birth) or hereditary factors resulting in weakened or deformed jaw bone



TREATMENT

HEALTH CARE

- Determined by the type of fracture, available equipment, supplies, and the veterinarian’s knowledge, experience, and comfort level
- Treatment selection is based on four major points: (1) reduction of the fracture to restore the bones and teeth (if possible) to their normal anatomic positions and reasonable contact of fracture ends; (2) re-establishment of the natural relationship or contact between the biting (incising) and chewing (masticatory) surfaces of the upper and lower teeth (occlusion), if possible; (3) stabilization sufficient for proper healing; (4) salvage condition (that is, the fracture cannot be repaired or stabilized sufficiently to allow for proper healing; therefore, treatment is designed to allow the animal to be functional, without restoring the bones and teeth to their normal positions)

HOME CARE

- Orthodontic wax—soft, pliable wax sent with owner to periodically cover any potentially irritating wires, which are used in repair of the fractured jaw
- Oral irrigation/hygiene products—use twice daily for oral hygiene and to reduce bacteria; chlorhexidine solutions help reduce bacteria; zinc and ascorbic acid solutions help reduce bacteria and stimulate soft-tissue healing

ACTIVITY

- Avoid hard chew items during healing process

DIET

- Soft food or gruel may be required during healing
- Nutritional and fluid maintenance required

SURGERY

- Treatment may involve a variety of methods to repair and/or stabilize the fractures; examples include use of a tape muzzle; wiring the jaws (maxilla and mandible) together; use of surgical pins to hold the fractured bone together; use of acrylic or composite splint; wiring around or between teeth; use of bone plates and screws
- Surgical procedures, such as removal of a portion of the jaw (examples are condylectomy and mandibulectomy), may be necessary for cases in which the fracture cannot be repaired or if massive injury is present—these are generally salvage procedures



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Pain Management

- Local anesthesia—nerve blocks to specific areas in the mouth (known as “intraoral local blocks”); regional nerve blocks: mental nerve, mandibular nerve, infraorbital nerve, and maxillary nerve
- Injectable pain relievers (analgesics)—butorphanol tartrate (Torbugesic®; Fort Dodge Animal Health); buprenorphine; nalbuphine
- Patches for pain relief—fentanyl (Duragesic®; Janssen)
- Oral pain relief medication (analgesics)—carprofen (Rimadyl®; Pfizer Animal Health); butorphanol tartrate (Torbugesic®); hydrocodone

Antibiotics

- Broad spectrum based on history, health, and chemical profile



FOLLOW-UP CARE

PATIENT MONITORING

- Physical examination—recheck 2 weeks after surgical repair
- X-rays—recheck 4 to 6 weeks after surgical repair, then every 2 weeks until fracture is healed and/or stabilizing appliance is removed
- Fracture site may temporarily (1 to 2 weeks) be more at risk to refracture after the support of the appliance is removed
- Once the fracture line is stable, compromised teeth may need additional treatment (such as a root canal) or careful extraction
- If the fracture healing process results in a deviation in the relationship or contact between the biting and chewing surfaces of the upper and lower teeth (malocclusion)—orthodontics, root canal or pulp capping, and/or selective extraction may be required
- Other considerations—stability of fracture and appliance; oral hygiene; oral intake of food and water; maintenance of weight; appropriate urination and defecation; indications of pain or swelling

PREVENTIONS AND AVOIDANCE

- Keep pet in an environment (such as a fenced yard, indoors) to minimize likelihood of trauma (such as being hit by a car)

POSSIBLE COMPLICATIONS

- Deviation in the relationship or contact between the biting and chewing surfaces of the upper and lower teeth (malocclusion)
- Disease involving the pulp, the tissue inside the hard portion of the tooth (known as “endodontic disease”)
- Inflammation/infection of the bone (osteomyelitis)
- Fractured bone does not heal (known as “nonunion” of the bone)
- A piece of bone that has separated from healthy tissue or does not have blood supply and dies (known as a “sequestrum”)
- Suture line splits open (known as a “dehiscence”)
- Nerve defects
- Facial pain
- Impaired chewing (mastication)

- Temporary weight loss
- Trauma to the soft tissues of the mouth (such as gums, tongue), due to appliance or wires

EXPECTED COURSE AND PROGNOSIS

- Generally good; however, predisposing factors, initiating force, location, type of fracture, quality of home care, and selection of treatment affect the healing outcome
- Usually takes 4 to 12 weeks to achieve bony union (that is, healing of the fracture)



KEY POINTS

- Fractures of the upper jaw (maxilla), the lower jaw (mandible), and associated structures are classified as to location, severity (that is, tooth involvement, soft-tissue tears, and type of bone fracture), and effects of the chewing muscles (muscles of mastication) on restoring the bones and teeth to their normal anatomic positions (reduction)
- Signs vary greatly according to the location, type, extent, cause of the fracture and underlying risk factors resulting in the injury
- Soft food or gruel may be required during healing
- Avoid hard chew items during healing process

TOOTH FRACTURE (BROKEN TOOTH)



BASICS

OVERVIEW

- Trauma to the tooth may involve fracture of any part of the tooth
- May involve the crown and root of the affected tooth; the crown of the tooth is the portion of the tooth that is above the gum that is covered by enamel; the root of the tooth is the part of the tooth below the gum line that is covered by cementum to attach the tooth to the bone of the jaw
- Classified as “uncomplicated” if the fracture does not enter the internal part of the tooth containing the blood vessels and nerves (known as the “pulp”) and “complicated” if the fracture enters the pulp

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Any age

SIGNS/OBSERVED CHANGES in the ANIMAL

Crown Fractures (involving the portion of the tooth above the gum line)

- Clinical loss of tooth-crown substance; may affect enamel only, or enamel and dentin; the enamel is the hard, shiny white material covering the crown of the tooth; the dentin makes up the bulk of the tooth structure
- Uncomplicated fractures with the fracture line close to the pulp (the internal part of the tooth, containing the blood vessels and nerves)—pale pink pulp is visible through the dentin; gentle exploring will not allow the explorer into the pulp cavity
- The recent or fresh complicated fracture is associated with bleeding from the pulp
- Older fractures may exhibit a dead pulp; clinically the pulp chamber is filled with dark material, and the tooth is often discolored

Root Fractures (involving the portion of the tooth below the gum line)

- May occur at any point along the root surface; often in combination with fracture of the crown, although root fractures can occur without fracture of the crown
- Root segments may remain aligned or may be displaced
- Clinical signs indicating a possible root fracture include pain on closure of the mouth or during open-mouth breathing
- Abnormal horizontal or vertical mobility of a tooth may raise suspicion of a root fracture

CAUSES

- Generally the result of a traumatic incident (such as a road traffic accident, blunt blow to the face, chewing on hard objects)



TREATMENT

HEALTH CARE

Uncomplicated Crown Fractures (involving the portion of the tooth above the gum line without entering the pulp)

- Dental procedure by the veterinarian in which sharp edges are removed with a bur and the exposed dentin tubules are sealed with a suitable liner or restorative material

Complicated Crown Fractures (involving the portion of the tooth above the gum line with entering the pulp)

- Require treatment of the internal part of the tooth containing the blood vessels and nerves, known as the “pulp;” such treatment is known as “endodontic therapy” and includes root canals and pulpotomy—if the tooth is to be maintained; otherwise, extraction is preferable to no treatment at all

Mature Tooth

- Recent fracture in the mature tooth with the pulp still alive (vital)—two options exist, partial pulpectomy and direct pulp capping (vital pulpotomy) followed by restoration or conventional root canal therapy and restoration
- For partial pulpectomy and direct pulp capping to succeed, the procedure should be carried out within hours of the injury; the initial

procedure may not be the final treatment—the tooth may require standard root canal treatment later, if the pulp tissue dies

- When the pulp already is inflamed chronically or is dead (known as “necrotic tissue”), standard root canal therapy and restoration are the treatments of choice, if the tooth is periodontally sound

Immature Tooth

- A living (vital) pulp is required for continued root development; as long as the pulp is alive (vital), the treatment of choice is partial pulpectomy and direct pulp capping, followed by restoration
- If the pulp tissue is dead (necrotic), no further root development will occur; necrotic immature teeth need endodontic treatment to be maintained; remove the dead tissue and pack the root canal with calcium hydroxide paste; some continued root development and closure of the apex can be stimulated if this procedure is performed; change the calcium hydroxide every 6 months until the apex is closed at which time a standard root canal is performed
- Immature teeth may be present in the mature animal, if trauma to the developing teeth caused death of the pulp; such teeth should be treated as “immature teeth”

Root Fractures (involving the portion of the tooth below the gum line)

- Treatment of crown and root fractures depends on how far below the gum line the fracture line extends
- If the fracture line does not involve the pulp (the internal part of the tooth, including the blood vessels and nerves) and does not extend more than 4 to 5 mm below the gum, restorative dentistry can be performed; if the fracture extends more than 5 mm below the gum and involves the pulp, the tooth usually should be extracted
- In some cases, the fractured tooth root may heal, if the tooth can be stabilized; in other cases, extraction of the tooth may be necessary



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- A broad-spectrum antibiotic drug for 5 to 7 days may be indicated; for example, when longstanding infection is present



FOLLOW-UP CARE

PATIENT MONITORING

- Check a partial pulpectomy and direct pulp-capping procedure with dental X-rays 6 and 12 months postoperatively, or at intervals determined by clinical signs, to detect death of the internal tissues of the tooth, including the blood vessels and nerves (pulp) and subsequent changes in the bone around the tips of the root, indicating the need for root-canal treatment
- Check the outcome of conventional root canal therapy by dental X-rays 6 to 12 months postoperatively; evidence of changes in the bone around the tips of the root at this time indicates the need for further treatment or extraction of the tooth; further treatment consists of redoing the root canal, often using surgical techniques
- Check root fractures with dental X-rays 6 to 12 months postoperatively
- Check uncomplicated fractures with dental X-rays at 4 to 6 months postoperatively

PREVENTIONS AND AVOIDANCE

- Avoid situations in which teeth are likely to be damaged; keep animal from chewing on hard objects, such as rocks
- To avoid complications, institute treatment within hours of injury

POSSIBLE COMPLICATIONS

- Untreated pulpal exposure invariably leads to inflammation of the pulp (known as “pulpitis”) and eventual death of the pulp tissue and subsequent changes in the bone around the tips of the root
- Immature teeth stop developing

EXPECTED COURSE AND PROGNOSIS

- Vary with vitality of the pulp (the internal structure of the tooth, including the blood vessels and nerves), location of the fracture, and whether the tooth is mature or immature



KEY POINTS

- Trauma to the tooth may involve fracture of any part of the tooth
- May involve the crown and root of the affected tooth; the crown of the tooth is the portion of the tooth that is above the gum that is covered by enamel; the root of the tooth is the part of the tooth below the gum line that is covered by cementum to attach the tooth to the bone of the jaw
- Avoid situations in which teeth are likely to be damaged; keep animal from chewing on hard objects, such as rocks
- To avoid complications, institute treatment within hours of injury

GASTRIC DILATATION-VOLVULUS (COMMONLY KNOWN AS “BLOAT”)



BASICS

OVERVIEW

- A disease in dogs in which the stomach dilates with gas and/or fluid (known as “gastric dilatation”), and subsequently rotates around its short axis (known as “volvulus”)
- Also known by the abbreviation, “GDV,” or by the term, “bloat”

GENETICS

- No direct genetic predisposition confirmed; however, dogs with a first-order relative with a history of GDV are at increased risk for development of GDV

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog; any large, deep-chested breed

Breed Predislection

- German shepherd dogs, Great Danes, standard poodles
- Rarely reported in smaller, deep-chested breeds, such as dachshunds and Pekingese

Mean Age and Range

- Any age; most commonly in middle-aged to older dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vomiting, which often progresses to “dry heaves”
- Anxious behavior
- Abdominal pain
- Abdominal distention; however, distended stomach may be contained under ribs, in which case abdominal distention may not be seen
- Collapse
- Drooling or excessive salivation (known as “ptyalism”)
- Depression
- Rapid heart rate (known as “tachycardia”)
- Rapid breathing (known as “tachypnea”) or difficulty breathing (known as “dyspnea”)
- Weak pulses; pale gums and moist tissues of the body (mucous membranes); and slowness of the pink color of the gums to return when the gums are blanched by finger pressure (known as “poor capillary refill time”) are suggestive of low blood volume (known as “hypovolemia”)

CAUSES

- Unknown
- Likely many factors, including anatomic (relating to the body structure), genetic and environmental factors

RISK FACTORS

- Classically has been linked to activity following a meal
- Anatomic predisposition in “deep-chested” dogs, particularly large- and giant-breed dogs
- Classically was thought that a lowered food bowl encouraged swallowing of air (known as “aerophagia”), which could lead to GDV; however, recent information identifies eating from a RAISED food bowl as a risk factor for development of GDV
- Having a first-degree relative with GDV and faster speed of eating have been identified as risk factors associated with the development of GDV
- Possibly having cancer in the gastrointestinal tract, as it can cause motility disturbances as well as retention of food and/or air in the stomach



TREATMENT

HEALTH CARE

- THIS DISEASE SYNDROME REPRESENTS AN EMERGENCY!

- Patients should be hospitalized, thoroughly assessed, and aggressively treated for poor circulation (known as “cardiovascular insufficiency”)
- Fluid therapy: crystalloid therapy, colloid therapy, or a combination can be used; crystalloids are fluids that contain electrolytes (chemical compounds, such as sodium, potassium, chloride) necessary for the body, crystalloids generally are similar to the fluid content (plasma) of the blood and move easily between the blood and body tissues, example is lactated Ringer’s solution; colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch
- Subsequent to stabilizing the circulation, decompression of the stomach should be performed
- Passing a stomach tube through the mouth, down the esophagus (the tube between the throat and the stomach), and into the stomach (known as “orogastric intubation”) is the preferred method of obtaining decompression of the stomach; commonly, considerable resistance is encountered upon reaching the area between the esophagus and the stomach, where the esophagus passes through the diaphragm (known as the “esophageal hiatus”)—the lubricated tube can be twisted or repositioned to facilitate passage of the stomach tube; differing patient positions (such as sitting, standing, lying down) can be attempted to facilitate passage
- In cases where passage of the stomach tube (orogastric intubation) is unsuccessful, passing a large-diameter sterile needle or catheter through the skin, abdominal wall, and into the stomach (known as “percutaneous gastrocentesis”) can be attempted; gas typically will make an audible noise when escaping—considerable time is necessary to achieve stomach decompression using this technique, owing to the small diameter of the needle in combination with the considerable amount of gas entrapped within the stomach
- Following patient stabilization and stomach decompression, surgical intervention is indicated

ACTIVITY

- Restriction of activity for approximately two weeks postoperatively is recommended

DIET

- Oral intake of food is recommended as soon as possible
- The role of food bowl height in the occurrence and recurrence of this disease is unclear at this time

SURGERY

- Surgical intervention should be performed as soon as possible in a stable patient or in a patient for which diligent stabilization efforts have proved to be ineffective
- Surgical intervention has three main goals: 1) returning the stomach (and spleen, if necessary) to its normal position in the body (known as “anatomical reposition”), 2) assessment of organ viability (in other words, “What is the status of the tissues?” “Are the tissues healthy enough to allow recovery and normal function?” “Are the tissues too deteriorated to allow recovery and can additional surgery salvage the organs?”), and 3) prevention of recurrence
- The stomach should be carefully and gently derotated back into its normal position in the body
- If the spleen has twisted or moved out of its normal position, it should be carefully and gently untwisted and moved to its normal position in the body
- Once repositioned, the stomach and spleen should be assessed; if areas are not healthy appearing or if obvious areas of dead tissue (known as “non-vital areas”) are present, surgical removal should be performed via removal of that part of the stomach (known as “partial gastrectomy”) that is unhealthy or dead and/or by removing the spleen (known as “splenectomy”)
- Prevention of recurrence is achieved through a permanent gastropexy (surgical attachment of the stomach to the abdominal wall); multiple techniques for performing gastropexy have been described and choice of technique is largely based on surgeon preference



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics are indicated around the time of surgery; depending on severity and progression of disease itself, surgery may be considered “clean,” “clean-contaminated,” “contaminated,” or “dirty”—it is often next to impossible to ascertain this information before the surgery is performed; these designations refer to findings of the status of the internal organs during surgery; the surgical procedure would be performed in a sterile surgery, but if the organs or tissues have died due to lack of adequate circulation, the surgeon may enter the

abdomen and find infection already present (so called, “dirty” surgery)

- Antibiotic selection should be based upon potential bacteria that may cause infection in the patient; moderate-to-severe disease may expose the dog to intestinal bacteria due to tearing of the gastrointestinal organs (known as “visceral perforation”) or loss of normal intestinal lining barriers to movement of bacteria from the gastrointestinal tract into the general circulation (known as “hematogenous bacterial translocation”); possible antibiotics include cefoxitin sodium and cefazolin sodium
- Agents to protect the stomach (known as “gastric protectants,” such as H₂-blockers [cimetidine, famotidine, ranitidine]) or coating agents (such as, sucralfate) may be implemented to minimize or prevent ulcers in the stomach and/or intestines



FOLLOW-UP CARE

PATIENT MONITORING

- General nursing care; some patients may require recumbent care for several days before eventual recovery
- Adequate pain control is necessary
- Abnormal or irregular heartbeats (known as “premature ventricular contractions”) commonly occur postoperatively in patients with GDV; these can be the result of poor blood flow to the heart muscle itself (known as “myocardial hypoperfusion”) and resultant damage, or due to damage to the spleen or removal of the spleen; monitoring of heart rhythm is recommended
- Correction of electrolyte abnormalities (particularly low levels of potassium in the blood [known as “hypokalemia”]) is often necessary
- Monitor urine production and kidney function postoperatively, particularly in patients that had sustained, significant low blood pressure (hypotension)

PREVENTIONS AND AVOIDANCE

- Elevation of food bowl is a matter of great debate; current literature suggests that elevating the food bowl actually may increase the risk of GDV; however, many owners/breeders continue to elevate food bowls
- Avoid strenuous exercise after eating or drinking
- Possibly slow down the rate of consumption of meals to reduce swallowing of air (aerophagia)
- Consider soaking dry food in water, before feeding or feed multiple, smaller meals

POSSIBLE COMPLICATIONS

- Gastric dilatation (stomach dilates with gas and/or fluid) may recur, even with successful surgical correction (known as “gastropexy”) performed; recurrence of volvulus (twisting or rotation of stomach) with an appropriately performed gastropexy technique is exceedingly rare
- Failure to remove dead stomach tissue may result in eventual stomach tearing (perforation) and bacterial infection of the lining of the abdomen (known as “septic peritonitis”)
- Irregular heartbeats (particularly premature ventricular complexes); blood-clotting disorder (known as “disseminated intravascular coagulopathy”); and ulcers of the stomach also may occur

EXPECTED COURSE AND PROGNOSIS

- Heightened awareness by dog owners combined with increased understanding by veterinarians of the complex events associated with GDV has reduced the mortality rate associated with this disease significantly over the past 30 years
- Prognosis in dogs treated appropriately that do not have dead stomach tissue (known as “gastric necrosis”) is excellent with a reported survival rate of 98%
- Dogs with dead stomach tissue (gastric necrosis) have a more guarded prognosis, with a reported survival rate of 66%



KEY POINTS

- Gastric dilatation-volvulus (“GDV” or “bloat”) is a potentially deadly disease that should be recognized and addressed immediately; failure to treat this disease quickly could lead to fatal consequences
- Initial efforts should be directed toward stabilizing the patient, as the disease often leads to severe circulatory problems
- Surgical intervention should be performed as soon as possible after initial stabilization
- Overall prognosis is fair, dogs without dead stomach tissue have a better prognosis than those with dead stomach tissue (gastric necrosis)

STOMACH (GASTRIC) MOTILITY DISORDERS

(ABNORMALITIES IN STOMACH EMPTYING)



BASICS

OVERVIEW

● Stomach (gastric) motility disorders result from conditions that directly or indirectly disrupt normal stomach emptying, which in turn may cause abnormal retention of food and fluid in the stomach (known as “gastric retention”), stomach distention, and subsequent signs, such as lack of appetite (anorexia), nausea, and vomiting

SIGNALMENT/DESCRIPTION of ANIMAL

Species

● Dogs and cats

Mean Age and Range

● Signs occur at any age, though it is uncommon to observe primary stomach (gastric) motility disorders in young animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs often are secondary to the primary cause of the stomach (gastric) motility disorder
- The major clinical sign is chronic vomiting of food following a meal (known as “postprandial vomiting”); the stomach normally should be empty after an average size meal in approximately 6 to 8 hours in dogs and 4 to 6 hours in cats (note: normal emptying times are influenced by meal volume, caloric density and fiber content); vomiting of undigested food greater than 10 to 12 hours following the meal suggests a stomach (gastric) motility disorder or blockage of the stomach or upper small intestine, preventing movement of the stomach contents out of the stomach (known as “outflow obstruction”)
- Vomiting can occur anytime following eating
- Distention of the stomach, nausea, lack of appetite (anorexia), belching, eating of nonfood items (known as “pica”), and weight loss
- Other signs and physical examination findings relate to the underlying cause of the disorder
- The veterinarian may detect decreased stomach sounds on listening to the abdomen with a stethoscope (known as “abdominal auscultation”)

CAUSES

- Primary stomach (gastric) motility disorders are often of unknown cause (known as “idiopathic gastric motility disorders”); they may arise from defects in normal electrical activity of muscle (known as “myoelectric activity”)
- Most motility disorders occur secondary to other primary conditions
- Metabolic disorders include low blood potassium levels (known as “hypokalemia”); excess levels of urea and other nitrogenous waste products in the blood (known as “uremia”); nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”); and inadequate levels of thyroid hormone (known as “hypothyroidism”)
- Nervous inhibition, as the result of stress, fear, pain or trauma
- Drugs, such as the anticholinergics (used as preanesthetics or to treat diarrhea, such as atropine); beta-blockers (used to treat heart and lung disease, such as isoproterenol), and narcotics
- Primary stomach disease, such as blockage of the stomach or upper small intestine, preventing movement of the stomach contents out of the stomach (outflow obstruction); inflammation of the stomach (gastritis); stomach ulcers; parvovirus infection
- Stomach surgery
- “Bloat” or gastric dilatation-volvulus syndrome (GDV)—a disease in dogs in which the stomach dilates with gas and/or fluid (known as “gastric dilatation”), and subsequently rotates around its short axis (known as “volvulus”)—is suspected to result from a primary motility disorder of abnormal muscle electrical and mechanical activity; dogs may continue to have signs of decreased stomach motility (known as “gastric hypomotility”) following surgical correction (known as a “gastropexy,” surgical attachment of the stomach to the abdominal wall)
- Backward or reverse flow of stomach contents into the esophagus (known as “gastroesophageal reflux”) and backward or reverse flow of intestinal contents into the stomach (known as “enterogastric reflux”) may result from decreased stomach motility (gastric hypomotility)
- Syndromes involving abnormal function of the autonomic nervous system (known as “dysautonomia syndromes”) have decreased stomach motility (gastric hypomotility) as part of a generalized disease process

RISK FACTORS

- Any potential stomach disease may result in secondary decreased motility (hypomotility)



TREATMENT

HEALTH CARE

- Most patients are treated as outpatients
- With severe vomiting or dehydration and electrolyte imbalance, hospitalization and specific therapy are necessary; electrolytes are chemical compounds, such as sodium, potassium, chloride, necessary for normal body function
- Dehydration with fluid and electrolyte imbalance requires appropriate fluid replacement

ACTIVITY

- Restrictions are based on the underlying disease

DIET

- Dietary manipulation is important in the management of primary stomach (gastric) motility disorders
- Diets should be formulated that are of liquid or semi-liquid consistency and low in fat and fiber content
- Small-volume meals, with frequent feeding, should be given
- Often dietary manipulation alone is successful in managing patients with delayed stomach (gastric) emptying from a motility disorder

SURGERY

- Dogs with chronic bloat (GDV) syndrome and retention of stomach contents (gastric retention) should have a surgical procedure (gastropexy) performed
- Following any stomach surgery, it may take as long as 14 days for motility to return to normal
- Patients with blockage of the stomach or upper small intestine, preventing movement of the stomach contents out of the stomach (“outflow obstruction”), require surgical correction



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Gastric Prokinetic Agents (drugs that improve the propulsion of contents through the stomach and into the intestines)

- Metoclopramide (Reglan®) improves stomach motility and coordinates stomach and upper small intestinal (duodenal) motility; also may prevent vomiting (known as an “antiemetic effect”)
- Cisapride works directly on gastrointestinal smooth muscle, stimulating motility; improves gastric emptying, and promotes increased motility of both the small and large intestine
- Tegaserod (Zelnorm®) is a newer prokinetic agent; it has similar prokinetic effects as cisapride; limited clinical experience in using this drug in the dog and cat
- Erythromycin given at low doses promotes stomach emptying
- H₂-blockers, such as ranitidine and nizatidine, have significant prokinetic effects on stomach motility similar to cisapride; neither cimetidine nor famotidine affects gastric emptying



FOLLOW-UP CARE

PATIENT MONITORING

- Response to therapy varies, according to the underlying cause of the stomach (gastric) motility disorder
- Failure to respond medically necessitates further investigation for mechanical obstruction

EXPECTED COURSE AND PROGNOSIS

- The length of treatment depends on the ability to resolve the underlying disorder or on response to therapy
- It may take stomach-surgery or parvovirus-infection cases 10 to 14 days to regain normal stomach function
- Cases involving generalized abnormal function of the autonomic nervous system (known as “generalized dysautonomia”) have grave prognoses



KEY POINTS

- Stomach (gastric) motility disorders result from conditions that directly or indirectly disrupt normal stomach emptying, which in turn may cause abnormal retention of food and fluid in the stomach (known as “gastric retention”), stomach distention, and subsequent signs, such as lack of appetite (anorexia), nausea, and vomiting
- Response to therapy varies, according to the underlying cause of the stomach (gastric) motility disorder
- Failure to respond medically necessitates further investigation for mechanical obstruction

CHRONIC GASTRITIS

(LONG-TERM INFLAMMATION OF THE STOMACH)



BASICS

OVERVIEW

- Intermittent vomiting of more than 1 to 2 weeks' duration, secondary to inflammation of the stomach (known as "gastritis")
- Presence of shallow ulcers (known as "erosions") or ulcers in the stomach, dependent on the cause and duration of the stomach inflammation

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Old, small-breed dogs (for example, Lhasa apso, shih tzu, miniature poodle)
- Basenjis and the Drentse patrijshond (Dutch partridge dog) breed can develop chronic hypertrophic gastritis, in which the stomach tissues are enlarged (known as "hypertrophy")

Mean Age and Range

- Varies with underlying cause

Predominant Sex

- Varies with underlying cause

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vomit is frequently bile stained, and may contain undigested food, flecks of blood, or digested blood (so called "coffee grounds")
- Frequency of vomiting varies from daily to every few weeks and increases as gastritis progresses
- Vomiting may be stimulated by eating or drinking
- Early morning vomiting before eating may indicate "bilious vomiting syndrome," a condition in which contents in the upper small intestine (duodenum) move backward into the stomach (known as "gastroduodenal reflux"); the contents contain bile acids, a normal product involved in fat digestion; the bile acids may interfere with the normal stomach lining protection and lead to inflammation of the stomach (gastritis)
- May see weight loss with long-term (chronic) loss of appetite (anorexia)
- May see black, tarry stools (known as "melena") due to the presence of digested blood in the bowel movement, if the animal has bleeding ulcers in the stomach
- Diarrhea, if animal also has intestinal disease
- May have pale gums and moist tissues of the body (known as "mucous membranes") if the animal has low red-blood cell counts (anemia) from long-term (chronic) blood loss

CAUSES

- Inflammatory—immune-mediated; dietary allergy or intolerance; unknown cause (known as "idiopathic" disease)
- Dietary indiscretion—animal eats plant material; foreign objects; chemical irritants
- Toxins—fertilizers; herbicides; cleaning agents; heavy metals
- Metabolic or endocrine (hormonal) disease—excess levels of urea and other nitrogenous waste products in the blood (known as "uremia" or "azotemia"); chronic liver disease; inadequate production of steroids by the adrenal glands (known as "hypoadrenocorticism" or "Addison's disease"); inflammation of the pancreas (known as "pancreatitis")
- Cancer—gastrinoma (a type of tumor usually found in the pancreas that secretes "gastrin," a hormone that stimulates acid production in the stomach); gastric adenocarcinoma (stomach cancer); gastrointestinal lymphoma (cancer originating from a type of white blood cell, known as a "lymphocyte," that develops in lymph nodes and other tissues of the body); leiomyosarcoma (cancer derived from smooth muscle of the stomach or intestines)
- Stomach polyps
- Parasitism—*Ollulanus tricuspis* and *Gnathostoma* (cats); *Physaloptera* (dogs, cats)
- Drugs—nonsteroidal anti-inflammatory drugs (NSAIDs); steroids
- Infectious—*Helicobacter*, bacteria associated with inflammation of the stomach (gastritis) and stomach ulcers; *Pythium*, a water mold that causes pythiosis; viral (canine distemper virus in dogs, feline leukemia virus [FeLV] in cats)

- Miscellaneous—backward or reverse flow of upper small intestinal (known as “duodenal”) contents into the stomach (known as “gastroduodenal reflux”) leading to bilious vomiting syndrome; stress; absence of hydrochloric acid (HCl) in the stomach fluids (known as “achlorhydria”)

RISK FACTORS

- Medications—nonsteroidal anti-inflammatory drugs (NSAIDs); steroids
- Environmental—unsupervised/free-roaming pets are more likely to ingest inappropriate foods or materials
- Ingestion of a dietary ingredient, to which an allergy or intolerance has been acquired



TREATMENT

HEALTH CARE

- Most patients are stable at presentation, unless vomiting is severe enough to cause dehydration
- Typically can be managed as an outpatient, pending diagnostic testing or undergoing clinical trials of special diets or medications
- If patient is dehydrated or if vomiting becomes severe, hospitalize and treat with appropriate intravenous (IV) fluid therapy

DIET

- “Nothing by mouth” (that is, no food or water by mouth) for 12 to 24 hours, if vomiting frequently
- Soft, low-fat food, ideally from single protein and carbohydrate sources
- Non-fat cottage cheese, skinless white-meat chicken, boiled hamburger or tofu as a protein source; and rice, pasta, or potato as a carbohydrate source, in a ratio of 1:3
- Frequent, small meals (every 4 to 6 hours or more frequently)
- Can use novel protein source (that is, feeding a protein to which the animal has never been exposed) or hydrolyzed protein diet (for which the protein source has been processed to break down the protein into smaller units, less likely to cause an allergic response), if dietary allergy is suspected
- Feed diets for a minimum of 3 weeks to assess adequacy of response; often requires longer trial periods of 6 to 8 weeks
- Feed a late night meal to help prevent bilious vomiting syndrome in the early morning hours

SURGERY

- Surgical management, if a mass or enlargement of stomach tissue (known as “hypertrophy”) is causing a blockage of the stomach, preventing movement of the stomach contents into the intestines (known as “outflow obstruction”)
- Surgical incision into the stomach (known as “gastrotomy”) for removal of foreign objects, if retrieval of the foreign object using a special, lighted, medical instrument (known as an “endoscope”) that is passed through the mouth and down the esophagus (the tube from the throat to the stomach) and into the stomach is unsuccessful or if an endoscope is not available



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Varies with underlying cause
- Treat any stomach erosions and ulcers
- Give steroids for long-term (chronic) inflammation of the stomach (gastritis) secondary to suspected immune-mediated mechanisms, if animal does not respond to dietary management
- Treatment for inflammation of the stomach caused by *Helicobacter*: amoxicillin, Pepto-Bismol® and metronidazole
- Medications to prevent or control vomiting (known as “antiemetics”) for fluid and electrolyte disorders caused by frequent or profuse vomiting; electrolytes are chemical compounds (such as sodium, potassium, chloride) necessary for the body to function
- Metoclopramide, cisapride, or low-dose erythromycin to increase stomach emptying and improve intestinal motility, if stomach emptying is delayed or backward or reverse flow of upper small intestinal (known as “duodenal”) contents into the stomach (known as “gastroduodenal reflux”) is present
- Synthetic prostaglandin E (misoprostol) to prevent stomach ulcers with nonsteroidal anti-inflammatory drug (NSAID) toxicity

- Drugs to suppress the immune response (known as “immunosuppressive drugs”), such as azathioprine, if an immune-mediated mechanism is suspected and the animal has not responded to dietary management and steroid administration adequately; expect response to occur in 2 to 3 weeks
- When in need of immediate suppression of the immune response (immunosuppression), use chlorambucil



FOLLOW-UP CARE

PATIENT MONITORING

- Resolution of clinical signs indicates a positive response
- Blood work to monitor electrolytes (such as sodium, potassium, and chloride) and acid–base status, if initially abnormal
- Complete blood counts should be obtained weekly and then reduced to every 4 to 6 weeks for patients on drugs to suppress the immune system—azathioprine, chlorambucil
- Repeat diagnostic workup and consider possible re-biopsy if signs decrease, but do not resolve

PREVENTIONS AND AVOIDANCE

- Avoid medications (such as steroids, nonsteroidal anti-inflammatory drugs [NSAIDs]) and foods that cause stomach irritation or allergic response in the patient
- Prevent free roaming of the animal and potential for dietary indiscretion

POSSIBLE COMPLICATIONS

- Progression of inflammation of the stomach (gastritis) from superficial (that is, involving just the surface of the lining) to atrophic gastritis (a condition in which the lining is thinner than normal)
- Stomach erosions and ulcers with progressive damage to the lining of the stomach (mucosal damage)
- Aspiration pneumonia
- Electrolyte or acid–base imbalances

EXPECTED COURSE AND PROGNOSIS

- Varies with underlying cause



KEY POINTS

- Inflammation of the stomach (gastritis) has numerous causes
- Diagnostic workup may be extensive; usually requires biopsy to identify disease (known as “definitive diagnosis”)

GASTRODUODENAL ULCER DISEASE

(ULCERS IN THE STOMACH AND UPPER SMALL INTESTINE [DUODENUM])



BASICS

OVERVIEW

●Ulcers in the stomach and upper small intestine (known as the “duodenum”) are called “gastroduodenal ulcers;” the ulcers extend through the lining (known as the “mucosa”) of the stomach and intestines and into the muscle layer of the stomach and intestines (known as the “muscularis mucosae”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

●Dogs and, less commonly, cats

Breed Predislection

●German Shepherd dogs are most susceptible to stomach ulceration after being given ibuprofen (NOTE: use of human over-the-counter pain relievers always should be discussed with your pet’s veterinarian prior to administration; these medications can cause serious side effects, such as stomach ulcers)

●Rottweilers have increased likelihood of tearing of the stomach and/or upper small intestine (duodenum) for unknown cause (known as “spontaneous gastroduodenal perforation”)

●Elite canine athletes have increased incidence of stomach ulceration, erosion (shallow ulcers) and/or bleeding during sustained strenuous exercise

●Chow chows appear to have an increased likelihood of stomach cancer (known as “gastric adenocarcinoma”)

Mean Age and Range

●All ages

Predominant Sex

●Male dogs have increased likelihood of having stomach cancer (known as “gastric carcinoma”)

SIGNS/OBSERVED CHANGES in the ANIMAL

●Some animals may not show any signs/changes, despite significant ulcers in the stomach or upper small intestine (duodenum)

●Vomiting: most common clinical sign

●Vomiting blood (known as “hematemesis”) may be present

●Black, tarry stools (known as “melena”), due to the presence of digested blood in the bowel movement, may be present

●Abdominal pain in the front of the abdomen (that is, near the rib cage)—patient may stand hunched over in the back or assume the “praying position”

●Lack of appetite (known as “anorexia”)

●Sluggishness (lethargy)

●Weight loss or extreme weight loss with muscle wasting (known as “cachexia”)

●Weakness, pale gums and moist tissues of the body, and/or collapse, if severe low red-blood cell count (anemia) or tearing (perforation) of the stomach or upper intestine with subsequent inflammation of the lining of the abdomen (known as “peritonitis”) develops

●Cats are less likely to show clinical evidence of bleeding from the stomach or upper small intestine (duodenum)

●Rapid heart rate (known as “tachycardia”); low blood pressure (known as “hypotension”); the pink color of the gums is slow to return when the gums are blanched by finger pressure (known as “prolonged capillary refill time”) if the animal has shock due to low volume of circulating blood (known as “hypovolemic shock”) or due to tearing (perforation) of the stomach or upper intestine (duodenum) with subsequent inflammation of the lining of the abdomen (peritonitis); high body temperature (known as “hyperthermia”)

●Abdominal distention may be present with tearing (perforation) of the stomach or upper intestine (duodenum) with subsequent inflammation of the lining of the abdomen (peritonitis)

●Fluid build-up in the tissues of the body (known as “edema”)—from blood/plasma loss causing low levels of protein in the blood (known as “hypoproteinemia”)

CAUSES

Drugs

●Nonsteroidal anti-inflammatory drugs (NSAIDs); steroids

Gastrointestinal Diseases

- Inflammatory bowel disease
- Cancer involving the mouth, esophagus (tube from the throat to the stomach), stomach or upper small intestine (duodenum)
- Foreign body in the mouth, esophagus, stomach or upper small intestine (duodenum)
- Correction of long-term (chronic) tear or abnormal opening in the diaphragm (known as “diaphragmatic hernia”)
- Excessive acid production by the stomach (known as “gastric hyperacidity”)
- Blockage of movement of stomach contents into the upper small intestine (known as “pyloric outflow obstruction”)

Infectious Diseases

- Parasites in the stomach and/or intestines
- *Pythium*, a water mold that causes “pythiosis”
- *Helicobacter*, bacteria associated with inflammation of the stomach (gastritis) and stomach ulcers
- Viral, fungal, or bacterial inflammation of the stomach and intestines (known as “gastroenteritis”)

Metabolic Diseases

- Kidney failure
- Liver failure
- Inadequate production of steroids by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”)
- Inflammation of the pancreas (known as “pancreatitis”)

Toxicity

- Heavy metal poisoning (such as arsenic, zinc, thallium, iron or lead)
- Plant intoxication (such as *Dieffenbachia*, sago palm, mushroom, castor bean)
- Chemical intoxication (such as phenol, ethylene glycol, corrosive agents, psoriasis creams—vitamin D analogues)
- Pesticide/rodenticide toxicity (such as cholecalciferol [vitamin D₃])

Cancer

- Mastocytosis (condition in which an abnormal number of mast cells are present in multiple tissues; mast cells contain histamine, and if it is released, it stimulates stomach-acid secretion)
- Gastrinoma (a type of tumor usually found in the pancreas that secretes “gastrin,” a hormone that stimulates acid production in the stomach)
- APUDoma (one of a group of tumor that produces hormones; a gastrinoma is one type of APUDoma)

Neurologic Diseases

- Head trauma
- Spinal cord disease

Stress/Major Medical Illness

- Generalized bacterial infection (known as “sepsis”)
- Shock
- Severe illness
- Burns
- Heat stroke
- Major surgery
- Trauma
- Low blood pressure (hypotension)
- Blood clots (known as “thromboembolic disease”)
- Sustained strenuous exercise

RISK FACTORS

- Administration of drugs known to cause ulcers of the stomach or upper small intestines—nonsteroidal anti-inflammatory drugs (NSAIDs) or steroids
- Critically ill patients
- Shock due to low circulating blood volume (hypovolemic shock) or due to generalized bacterial infection (known as “septic shock”)
- Pythiosis has a regional distribution—states that border the Gulf of Mexico



TREATMENT

HEALTH CARE

- Treat any underlying causes
- The veterinarian may use a special lighted instrument, called an “endoscope” (a lighted medical instrument that is passed through the mouth into the esophagus [the tube from the throat to the stomach] and into the stomach and possibly the upper small intestine [duodenum]) to evaluate the ulcers and/or bleeding
- Treat on an outpatient basis if the cause is identified and removed, vomiting is not excessive, and bleeding is minimal in the stomach and/or upper small intestine (duodenum)
- Inpatient treatment for severe bleeding in the stomach and/or upper small intestine (duodenum) and/or tearing of the stomach or upper small intestine at the site of the ulcer (known as “ulcer perforation”); excessive vomiting; and/or shock
- May need emergency management of bleeding, shock or bacterial infection/inflammation of the lining of the abdomen (known as “septic peritonitis”)
- Intravenous fluids to maintain hydration, to maintain blood flow to the lining of the stomach, and/or to treat shock
- May need transfusions (whole blood or packed red-blood cells) or oxygen-carrying hemoglobin solution infusions in patients with severe bleeding in the stomach and/or upper small intestine (duodenum)
- Patients with very low levels of protein in their blood (known as “hypoproteinemia”) may require colloids and/or plasma to improve blood volume; colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch
- In severe cases of vomiting blood—to stop the bleeding into the stomach and/or upper small intestine (duodenum), ice water flush (lavage) or lavage with norepinephrine diluted in ice water can be attempted

ACTIVITY

- Restricted

DIET

- Discontinue intake of food and/or water by mouth, if vomiting
- When feeding is resumed, feed small amounts in multiple feedings.
- Recommended highly digestible diet with low- to moderate-fat (high dietary fat delays stomach emptying) and low-fiber content. Dependent on the cause of the ulcers in the stomach or upper small intestine (duodenum), diet with a novel protein source (that is, feeding a protein to which the animal has never been exposed) or hydrolyzed protein diet (where the protein source has been processed to break down the protein into smaller units, less likely to cause an allergic response) may be beneficial

SURGERY

- Surgical treatment is indicated if medical treatment fails after 7 to 10 days; bleeding is uncontrolled and severe; tearing of the stomach or upper small intestine at the site of the ulcer (ulcer perforation); and/or a tumor is identified that potentially could be removed surgically



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Histamine (H_2) blockers competitively inhibit stomach acid secretion and are the initial drug of choice (such as cimetidine, ranitidine, famotidine, nizatidine); H_2 -blockers differ in potency and duration of action—famotidine is most potent, followed by ranitidine and then cimetidine—treat for at least 6 to 8 weeks; rebound excessive production of stomach acid may occur when H_2 -blockers are discontinued, but can be minimized by tapering the dose as it is discontinued
- Antacids neutralize stomach acid and some induce local production of compounds that protect the lining of the stomach and upper small intestine (known as “mucosal protectants”), but must be given at least four to six times per day to be effective
- Sucralfate suspension protects ulcerated tissue by binding to ulcer sites and stimulating production of prostaglandins; binding is greater in upper small intestinal (duodenal) ulcers than in stomach ulcers
- Antibiotic(s) should be given by injection, if a break in the lining of the stomach or upper small intestine is suspected or if aspiration

pneumonia is present

- Drugs to stop or control vomiting (known as “antiemetics,” such as chlorpromazine and prochlorperazine) are administered if vomiting occurs frequently or results in significant fluid losses
- Omeprazole—most potent inhibitor of secretion of stomach acid; treatment of choice for gastrinomas (tumors usually found in the pancreas that secrete “gastrin,” a hormone that stimulates acid production in the stomach) with evidence of spread (that is, metastasis) or that cannot be removed surgically (known as “nonresectable disease”) and disease of the stomach and upper small intestine (duodenum) that has not responded to H₂-blocker therapy
- Misoprostol, a synthetic prostaglandin analogue, may decrease the secretion of stomach acid and also protect the lining of the stomach and upper small intestine; helps prevent and treat nonsteroidal anti-inflammatory drug (NSAID)-induced ulcers; it may have some effect in treating ulcers of the stomach and upper small intestine (duodenum) from other causes



FOLLOW-UP CARE

PATIENT MONITORING

- Improvement may be assessed on resolution of clinical signs; monitoring packed cell volume and total protein (blood tests that determine the volume of red blood cells as compared to the fluid portion of the blood and the concentration of protein in the blood, respectively); fecal occult blood (in which the presence or absence of blood in the bowel movement is evaluated); and blood urea nitrogen or “BUN” (blood test that indicates the level of urea in the blood—elevated urea levels can be a sign of dehydration) may help to detect continued blood loss and dehydration
- Repeat evaluation using an endoscope is recommended for advanced cases to help determine appropriate duration of therapy
- Depending on the underlying cause, specific laboratory or imaging tests (such as X-rays, contrast X-rays, ultrasound examination) may be necessary to monitor response to therapy

PREVENTIONS AND AVOIDANCE

- Avoid gastric irritants (such as nonsteroidal anti-inflammatory drugs [NSAIDs]; steroids)
- Use misoprostol when treating with NSAIDs, to protect the lining of the stomach and upper small intestine
- Administer NSAIDs with food, to try to minimize irritation of the stomach and upper small intestine
- COX-2 selective or dual LOX/COX inhibitors may have less adverse effects on the stomach and upper small intestine than nonselective NSAIDs

POSSIBLE COMPLICATIONS

- Severe blood loss requiring transfusion
- Generalized bacterial infection (sepsis)
- Tearing of the stomach or upper small intestine at the site of the ulcer (ulcer perforation)
- Death
- Aspiration pneumonia—rare

EXPECTED COURSE AND PROGNOSIS

- Varies with underlying cause
- Patients with stomach cancer, kidney failure, liver failure, pythiosis, systemic mastocytosis, generalized bacterial infection (sepsis), and/or gastric perforation—prognosis is guarded to poor
- Ulcers involving the stomach and/or upper small intestine (duodenum) secondary to nonsteroidal anti-inflammatory drug (NSAID) administration, inflammatory bowel disease, or inadequate production of steroids by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”)—prognosis may be good to excellent, depending on severity of disease



KEY POINTS

- Ulcers in the stomach and upper small intestine (known as the “duodenum”) are called “gastroduodenal ulcers;” the ulcers extend through the lining (known as the “mucosa”) of the stomach and intestines and into the muscle layer of the stomach and intestines (known as the “muscularis mucosae”)
- Nonsteroidal anti-inflammatory drugs (NSAIDs) should be administered to pets only under the guidance of a veterinarian
- Administration of NSAIDs can result in ulceration and tearing (perforation) of the stomach and upper small intestine (duodenum)

- Adverse effects of NSAIDs can be reduced by giving drug with food and giving a synthetic prostaglandin analogue (such as misoprostol) to protect the lining of the stomach and upper small intestine (duodenum)

EOSINOPHILIC GASTROENTERITIS

(INFLAMMATION OF THE STOMACH AND INTESTINES, CHARACTERIZED BY THE PRESENCE OF EOSINOPHILS [A TYPE OF WHITE BLOOD CELL])



BASICS

OVERVIEW

- An inflammatory disease of the stomach and intestine (generally known as “gastroenteritis”), characterized by an infiltration of eosinophils (a type of white blood cell), usually into the lamina propria (the layer just under the lining), but occasionally involving deeper tissues, known as the “submucosa” (the layer of tissue between the lining and the muscular layer of a tubular organ) and “muscularis” (the muscular layer of a tubular organ)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog and cat; reportedly more common in dogs than in cats

Breed Predilections

- German shepherd dog, rottweiler, soft-coated wheaten terrier, and Chinese shar pei may be more likely to have eosinophilic gastroenteritis than other breeds

Mean Age and Range

- Dogs—most common in animals less than 5 years of age, although any age may be affected
- Cats—median age, 8 years; range, 1.5 to 11 years reported

SIGNS/OBSERVED CHANGES in the ANIMAL

- Intermittent vomiting, small-bowel diarrhea, lack of appetite (anorexia), and/or weight loss are most common; signs are similar to those seen with other causes of gastroenteritis
- One report states that 50% of cats with eosinophilic inflammation of the stomach (known as “gastritis”) and/or inflammation of the intestines (known as “enteritis”) had blood in the stool (known as “hematochezia”) or black, tarry stools (known as “melena”) due to the presence of digested blood in the bowel movement
- Cats—thickened bowel loops may be palpated by the veterinarian during physical examination
- If hypereosinophilic syndrome (a condition in which the animal has a high eosinophil [type of white blood cell] count due to overproduction of eosinophils by the bone marrow; the eosinophils invade various tissues [including the gastrointestinal tract] and cause organ damage) is the cause of the inflammation in the stomach and/or intestines, enlarged lymph nodes, liver enlargement, and/or spleen enlargement also may be noted on physical examination

CAUSES

- Unknown cause (known as “idiopathic eosinophilic gastroenteritis”)
- Parasitic disease
- Immune-mediated disease—food allergy; adverse drug reaction; associated with other forms of inflammatory bowel disease
- Systemic mastocytosis (condition in which an abnormal number of mast cells are present in multiple tissues; mast cells contain histamine, and if it is released, it stimulates stomach-acid secretion)
- Hypereosinophilic syndrome (a condition in which the animal has a high eosinophil [a type of white blood cell] count due to overproduction of eosinophils by the bone marrow; the eosinophils invade various tissues [including the gastrointestinal tract] and cause organ damage)
- Eosinophilic leukemia (type of blood cancer in which the abnormal cells are eosinophils [a type of white blood cell])
- Eosinophilic granuloma (a mass or nodular lesion containing a type of white blood cell, called an eosinophil)



TREATMENT

HEALTH CARE

- Most can be treated successfully on an outpatient basis
- Patients with systemic mastocytosis (condition in which an abnormal number of mast cells are present in multiple tissues; mast cells

contain histamine, and if it is released, it stimulates stomach-acid secretion); disease in which protein is lost from the body into the intestinal tract (known as “protein-losing enteropathy”), or other illnesses occurring at the same time as the eosinophilic gastroenteritis may require hospitalization until they are stabilized

- If the patient is dehydrated or must be withheld from food or water (that is “nothing by mouth” or “NPO”) because of vomiting, fluids (such as lactated Ringer’s solution) should be administered
- If the animal has severely low levels of albumin (a type of protein) in the blood (known as “hypoalbuminemia”) from protein-losing enteropathy, consider administration of colloids—colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch

ACTIVITY

- No need to restrict, unless severely debilitated

DIET

- Dietary manipulation is usually a critical component of therapy
- Highly digestible diets with limited nutrient sources (known as “hypoallergenic diets”)—extremely useful for eliciting remission; can be used as maintenance diets once the patient is stabilized; most cases are managed successfully using dietary manipulation
- Dog—diet examples include: Hill’s Prescription Diet® d/d®, z/d®, and i/d®; Purina Veterinary Diets® EN GastroENteric® brand Canine Formula, HA HypoAllergenic® brand Canine Formula, and LA Limited Antigen® brand Canine Formula; Royal Canin™/Innovative Veterinary Diets Limited Protein diets; Eukanuba® Low Residue™ Diet and Response™ Formula FP or KO; balanced homemade diets
- Cat—diet examples include: Hill’s Prescription Diet® i/d®, z/d®, m/d® and d/d®; Purina Veterinary Diets® DM Diabetes Management® brand Feline Formula and EN GastroENteric® brand Feline Formula; Royal Canin™/Innovative Veterinary Diets Limited Protein diets; Eukanuba® Low Residue™ Diet
- Monomeric diets are designed to provide protein as either peptides or amino acids (the smallest component of protein); these diets require little digestion—they have “nonallergenic” components; can be used in patients that are not vomiting, but have moderate-to-severe inflammation of the stomach and intestines (gastroenteritis); useful if a food allergy is suspected
- Patients with severe intestinal involvement and significant loss of protein through the intestines (protein-losing enteropathy), may need intravenous feeding (known as “total parenteral nutrition” or “TPN”) until remission of disease is obtained; it is rare that TPN is necessary
- Once the patient is stabilized, an elimination-diet trial may be instituted, if food allergy or intolerance is the suspected cause, to attempt to identify the offending nutrient



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Corticosteroids—mainstay of treatment; prednisone used most frequently
- Gradually taper dose of corticosteroids, following your pet’s veterinarian’s recommendations; relapses are more common in patients that are taken off corticosteroids too quickly
- Occasionally other drugs that suppress or decrease the immune response (known as “immunosuppressive drugs”) can be used to allow a reduction in corticosteroid dose and avoid some of the adverse effects of steroid therapy; these immunosuppressive medications also may be added in cases where the animal does not respond or responds poorly to dietary modification and medication
- Azathioprine is the most common additional immunosuppressive therapy
- Chlorambucil can be used as an additional immunosuppressive therapy
- Budesonide, a new oral corticosteroid, has been used to treat cats and dogs with inflammatory bowel disease successfully; it has been approved recently in the United States, but must be compounded (that is, prepared) for use in animals by a pharmacist



FOLLOW-UP CARE

PATIENT MONITORING

- Initially frequent for more severely affected patients; doing blood counts to determine eosinophil counts may be helpful; the corticosteroid dosage usually is adjusted during these visits
- Patients with less severe disease may be checked 2 to 5 weeks after the initial evaluation; monthly to bimonthly thereafter until

corticosteroid therapy is completed

- Patients receiving azathioprine or chlorambucil—complete blood count (CBC) should be performed every 10 to 14 days after the start of treatment, with rechecks monthly and then bimonthly thereafter for the entire treatment period; bone-marrow suppression, leading to low red-blood cell and low white-blood cell counts, can be seen at any time during treatment and generally is reversible with drug discontinuation
- Patients usually do not require long-term follow-up, unless the problem recurs

PREVENTIONS AND AVOIDANCE

- If a food allergy or intolerance is suspected or documented, avoid feeding that particular nutrient and adhere strictly to dietary changes recommended by your pet's veterinarian

POSSIBLE COMPLICATIONS

- Weight loss, debilitation in cases that do not respond or respond poorly to dietary manipulation or medication
- Adverse effects of corticosteroid therapy, such as excessive thirst (known as “polydipsia”) and excessive urination (known as “polyuria”)
- Bone-marrow suppression (leading to low red-blood cell and low white-blood cell counts); inflammation of the pancreas (known as “pancreatitis”); inflammation of the liver (known as “hepatitis”); or lack of appetite (anorexia) caused by azathioprine and/or chlorambucil

EXPECTED COURSE AND PROGNOSIS

- Majority of dogs with eosinophilic gastroenteritis respond to a combination of dietary manipulation and steroid therapy
- Cats often have more severe disease, with a poorer prognosis than dogs
- Cats often require higher doses of corticosteroids for longer periods of time to elicit remission



KEY POINTS

- Eosinophilic gastroenteritis tends to be a waxing and waning disease, meaning that the animal may have signs for a while and then have a period without signs and then the signs recur
- Need vigilance for the life of the pet regarding inciting factors, such as avoiding certain food ingredient
- Potential that long-term therapy will be needed

HEMORRHAGIC GASTROENTERITIS

(SUDDEN, BLOODY INFLAMMATION OF THE STOMACH AND INTESTINES)



BASICS

OVERVIEW

- Very sudden (known as “peracute”) bloody inflammation of the intestines (known as “hemorrhagic enteritis”) of dogs, characterized by a sudden onset of severe bloody diarrhea that is often explosive; the dog also has vomiting (therefore, the disease is named “hemorrhagic gastroenteritis”), low circulating blood volume (known as “hypovolemia”) and marked increase in the percentage volume of red-blood cells as compared to the fluid volume of blood (known as “hemoconcentration”) due to a dramatic loss of water and electrolytes (chemical compounds, such as sodium, potassium, chloride, necessary for the body to function) into the intestinal lumen
- Also known as “HGE”

GENETICS

- Unknown; however, specific small-breed dogs may be more likely to develop hemorrhagic gastroenteritis than other breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- All breeds can be affected, but the incidence is greater in small-breed dogs; breeds most represented include miniature schnauzers, dachshunds, Yorkshire terriers, and miniature poodles

Mean Age and Range

- Usually seen in adult dogs, with a mean age of 5 years

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs are variable in both the course and severity of the disease; the disease usually is very sudden (peracute) and associated with shock due to low circulating blood volume (known as “hypovolemic shock”)
- Most animals affected have been healthy prior to having signs, with no historical environmental changes or other ongoing disease involving the stomach and/or intestines
- Signs usually begin with sudden (acute) vomiting, lack of appetite (anorexia), and depression that then is followed by watery diarrhea, quickly changing to bloody diarrhea
- Signs progress rapidly and become severe within a period of hours (usually 8 to 12 hours) and are the result of shock due to low circulating blood volume (hypovolemic shock) and marked increase in the percentage volume of red-blood cells as compared to the fluid volume of blood (hemoconcentration) due to a dramatic loss of water and electrolytes (chemical compounds, such as sodium, potassium, chloride, necessary for the body to function) into the intestinal lumen
- The patient generally is depressed and weak and has prolonged capillary refill time (that is, the pink color of the gums is slow to return when the gums are blanched by finger pressure) and weak pulse pressure
- Skin turgor (turgor is the normal fullness or tension of tissues resulting from fluid content) may appear normal due to the very sudden (peracute) nature of the disease and the lag time in body fluids moving from the skin tissues into the central organs (known as “compartmental shifts”), so that the skin turgor does not reflect the animal’s dehydration
- The abdomen may be painful when the veterinarian feels it (known as “abdominal palpation”) and s/he may feel fluid-filled intestines
- Rectal examination will identify bloody diarrhea, and later in the course of disease, a “raspberry jam” characteristic stool develops
- Occasionally fever, but often body temperature is normal or even subnormal

CAUSES

- Unknown
- Type 1 hypersensitivity reaction (immune reaction) directed against the dog’s intestinal lining
- Bacterial cultures of some dogs with HGE yield mostly pure cultures of a bacteria, *Clostridium perfringens*, and its related intestinal poison (known as an “enterotoxin”), but the significance of these findings is unknown
- Searches for poison-producing (known as “toxigenic”) *E. coli* strains have been unrewarding

RISK FACTORS

- Unknown

- Most dogs are previously healthy with no major ongoing illness



TREATMENT

HEALTH CARE

- Patients suspected of having acute HGE should be hospitalized and treated aggressively, because clinical deterioration is often rapid and can be fatal
- Rapid fluid-volume replacement is required
- Intravenous (IV) fluids containing balanced electrolyte solutions are given rapidly until the packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood) is less than 50%; electrolytes are chemical compounds (such as sodium, potassium, chloride) necessary for the body to function
- A moderate rate of maintenance fluids is given to maintain circulatory function and to correct any potassium or other electrolyte deficits during the recovery period
- Continued body-fluid losses through the stomach (by vomiting) or intestines (by vomiting and/or diarrhea) should be estimated and that volume added to the fluid requirements
- Animals with low levels of protein in their blood (known as “hypoproteinemia”) may require treatment with colloids or plasma; colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch

ACTIVITY

- Restricted

DIET

- No food or drink by mouth (known as “NPO”) during acute disease
- During recovery period, feed a bland, low-fat, low-fiber diet for several days before returning to the normal diet



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Injectable antibiotics are given because of the potential for the spread of bacteria and their poisons in the blood (known as “blood poisoning” or “septicemia”) and possible implications of the bacteria, *Clostridium perfringens*, being involved in the disease; ampicillin is recommended
- Alternate antibiotic choices include trimethoprim-sulfa or cephalosporins; ampicillin in combination with gentamicin or a fluoroquinolone (such as, enrofloxacin) is suggested in cases of suspected septicemia
- Short-acting steroids have been suggested by some, reasoning that a hypersensitivity reaction may be involved; for example, dexamethasone sodium phosphate
- Excessive blood loss may require a blood transfusion (rare)
- Antibiotics by mouth and intestinal protectants are of little benefit, and generally not administered
- Rectal administration of agents to protect the lining of the intestines (known as “mucosal protectants”) is of questionable value
- Drugs to control vomiting (known as “antiemetics”) may be given for severe vomiting
- Drugs that change the motility of the intestines (known as “intestinal motility modifiers”) are not considered necessary and are not recommended



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor the packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid

volume of blood) and total solids (a quick laboratory test that provides general information on the level of protein in the fluid portion of the blood) frequently (at least every 4 to 6 hours)

- Modify fluid replacement based on PCV, continued fluid losses from the stomach and/or intestines, and circulatory function
- If clinical improvement is not seen in 24 to 48 hours, reevaluate the patient, as other causes of hemorrhagic diarrhea are probable

POSSIBLE COMPLICATIONS

- Occasionally a blood-clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”) may develop
- Nervous system signs or even seizures may occur secondary to “sludging of the blood”—the very high percentage volume of red-blood cells compared to the fluid volume of blood (hemoconcentration) makes it difficult to move the blood through the blood vessels
- Irregular heart beats and rhythms may occur from suspected myocardial reperfusion injury (a condition that may occur when the heart has had poor blood flow or circulation with low levels of oxygen and then the blood flow and oxygenation is restored; the previously oxygen-starved heart muscle may release high levels of free radicals, which causes more heart muscle tissue damage; blood flow is known as “perfusion” and the reestablishment of blood flow is known as “reperfusion”)
- A hemolytic-uremic syndrome (a syndrome in which the red-blood cells break down [known as “hemolysis”] and excess levels of urea and other nitrogenous waste products build up in the blood [known as “uremia”]) may occur (rare)

EXPECTED COURSE AND PROGNOSIS

- Course of the disease is generally short, lasting from 24 to 72 hours
- Prognosis is good, and most patients recover with no complications
- Sudden death is uncommon.



KEY POINTS

- Very sudden (known as “peracute”) bloody inflammation of the intestines (known as “hemorrhagic enteritis”) of dogs, characterized by a sudden onset of severe bloody diarrhea that is often explosive; the dog also has vomiting (therefore, the disease is named “hemorrhagic gastroenteritis”), low circulating blood volume (known as “hypovolemia”) and marked increase in the percentage volume of red-blood cells as compared to the fluid volume of blood (known as “hemoconcentration”) due to a dramatic loss of water and electrolytes into the intestinal lumen
- Immediate and aggressive medical management is needed
- With appropriate therapy, mortality is usually low
- Recurrence is reported in about 10% of cases

LYMPHOPLASMACYTIC-PLASMACYTIC GASTROENTERITIS (INFLAMMATION OF THE STOMACH AND INTESTINES, CHARACTERIZED BY THE PRESENCE OF LYMPHOCYTES AND PLASMACYTES [TYPES OF WHITE-BLOOD CELL])



BASICS

OVERVIEW

- An inflammatory disease of the stomach and intestine (generally known as “gastroenteritis”), characterized by infiltration of lymphocytes (a type of white-blood cell) and plasma cells or plasmacytes (a specialized type of white-blood cell; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody, necessary for fighting disease); the lymphocytes and plasma cells usually infiltrate into the lamina propria (the layer just under the lining), but occasionally involve deeper tissues, known as the “submucosa” (the layer of tissue between the lining and the muscular layer of a tubular organ) and “muscularis” (the muscular layer of a tubular organ)
- Most common form of inflammatory bowel disease (IBD) affecting dogs and cats

GENETICS

- Basenjis, Norwegian lundehunds, and soft-coated wheaten terriers have particular familial forms of inflammatory bowel disease (IBD)
- Certain genes may make an individual susceptible to development of IBD

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog and cat

Breed Predilections

- Basenjis and Norwegian lundehunds have particular forms of inflammatory bowel disease (IBD); gluten-sensitive enteropathy (specific type of intestinal disease related to the presence of wheat gluten in the diet) affects Irish setters; protein-losing enteropathy and nephropathy (conditions in which proteins are lost from the body through the intestines [enteropathy] or kidneys [nephropathy]) affects soft-coated wheaten terriers
- German shepherd dogs and Chinese shar peis reportedly are susceptible to lymphocytic-plasmacytic gastroenteritis
- Pure-breed cats may be more likely to have lymphocytic-plasmacytic gastroenteritis than other cats

Mean Age And Range

- Most common in middle-aged animals
- Dogs as young as 8 months and cats as young as 5 months of age have been reported to have lymphocytic-plasmacytic gastroenteritis

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs associated with lymphocytic-plasmacytic inflammation of the stomach (known as “gastritis”) with or without inflammation of the intestines (known as “enteritis”) can vary in type, severity, and frequency
- Generally have an intermittent, long-term (chronic) course, but may increase in frequency over time
- Cats—intermittent, chronic vomiting is the most common sign; long-term (chronic) small-bowel diarrhea is second most common sign
- Dogs—long-term (chronic) small-bowel diarrhea is the most common sign; if only the stomach is involved, vomiting is the most common sign
- Dogs and cats—lack of appetite (anorexia) and long-term (chronic) weight loss are common; while blood in the stool (known as “hematochezia”); vomiting blood (known as “hematemesis”); and dark, tarry stools (known as “melena”) due to the presence of digested blood in the bowel movement are noted occasionally
- Animal may have normal body-fluid status (that is, normal hydration) or may have low body-fluid status (that is, dehydration); may have extreme weight loss with muscle wasting (known as “cachexia”), and may show signs of depression, depending on the disease severity and organ(s) affected

CAUSES

- Probably many factors cause lymphocytic-plasmacytic gastroenteritis; may involve changes in the bacteria found in the intestinal tract and in the immune response

Infectious Agents

- *Giardia*, *Salmonella*, *Campylobacter*, and normal resident gastrointestinal bacteria have been implicated in causing

lymphocytic-plasmacytic gastroenteritis, but not proven

Dietary Agents

- Meat proteins, food additives, artificial coloring, preservatives, milk proteins, and gluten (wheat) have been proposed as causes

Genetic Factors

- Certain forms of inflammatory bowel disease (IBD) are more common in some breeds of dogs
- Certain genes may make an individual susceptible to development of IBD



TREATMENT

HEALTH CARE

- Outpatient, unless the patient is debilitated from dehydration, has low levels of protein in the blood (known as “hypoproteinemia”), or has extreme weight loss with muscle wasting (cachexia)
- If the patient is dehydrated or must have food and water withheld because of severe vomiting, fluid therapy (such as lactated Ringer’s solution) should be administered; additional supplementation of certain compounds (known as ‘electrolytes,’ such as potassium chloride, magnesium sulfate) may be necessary if abnormalities in levels in the blood are detected
- Colloids (dextrans or hetastarch) should be given if severely low levels of albumin (a protein) are present in the blood (known as “hypoalbuminemia”) from protein-losing enteropathy (condition in which proteins are lost from the body through the intestines) is present; colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume

ACTIVITY

- No restrictions

DIET

- Dietary therapy is an essential component of patient management
- Patients with severe intestinal involvement and protein-losing enteropathy (condition in which proteins are lost from the body through the intestines) may require intravenous feeding (known as “total parenteral nutrition” or “TPN”) until stable
- Highly digestible, antigen-restricted (so-called “low-allergy”) diets, containing a single protein source, should be fed to eliminate the possibility of food allergy
- Highly digestible diets decrease the intestinal antigenic load, thus helping to reduce inflammation of the lining of the intestines; appropriate diet therapy can contribute to remission of signs and can be used as a maintenance diet
- Modification of the n-3 to n-6 fatty-acid ratio may help to modulate the inflammatory response
- Injectable cobalamin (vitamin B12) supplementation is essential if serum levels are subnormal, as deficiencies can contribute to clinical signs and limit the effectiveness of dietary and medical therapy
- Numerous commercial elimination diets (diets that eliminates or excludes the food ingredient(s) to which the animal is allergic or intolerant) are available for dogs and cats; home-cooked diets also are an excellent option, but are more time consuming for owners



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Corticosteroids—mainstay of treatment for lymphocytic-plasmacytic inflammation of the intestines of unknown cause (known as “idiopathic lymphocytic-plasmacytic enteritis”); prednisone used most frequently in dogs and cats; cats may require higher dosages to control their disease and may respond better to prednisolone than to prednisone
- Budesonide, a locally active steroid, may be used in patients that cannot tolerate the systemic side effects of prednisone, such as excessive thirst (known as “polydipsia”) and excessive urination (known as “polyuria”)
- Injectable steroids may be needed in severe cases, in which absorption of the drug following dosage by mouth may be limited
- Gradually taper dose of corticosteroids, following your pet’s veterinarian’s recommendations; relapses are more common in patients that are taken off corticosteroids too quickly; maintenance dosages of steroids, administered every 48 to 72 hours may be necessary in some patients
- Occasionally other drugs that suppress or decrease the immune response (known as “immunosuppressive drugs”) can be used to allow a reduction in corticosteroid dose and avoid some of the adverse effects of steroid therapy
- Azathioprine—an immunosuppressive drug that can be used to allow a reduction in corticosteroid dose; delayed onset of activity (up to

three weeks) limits effectiveness in sudden (acute) disease

- Chlorambucil is an effective alternative to azathioprine
- Metronidazole—has antibacterial and antiprotozoal properties; some evidence that it also has immune-modulating effects
- Cyclosporine—may be useful in the therapy of animals that do not respond or respond poorly to dietary modification and other medications; using Neoral® or Atopica®; dosage is very individualized, so monitoring is recommended; cost prohibits routine use of this drug
- Sulfasalazine—a sulfa analog that is broken down by intestinal bacteria into sulfapyridine and 5-aminosalicylic acid, the later of which provides anti-inflammatory effects in the colon or large bowel



FOLLOW-UP CARE

PATIENT MONITORING

- Severely affected patients on bone-marrow suppressive medications require frequent monitoring; adjust medications during these visits based on blood work and clinical signs
- Patients receiving azathioprine or chlorambucil—complete blood count (CBC) should be performed every 10 to 14 days after the start of treatment, with rechecks monthly and then bimonthly thereafter for the entire treatment period; bone-marrow suppression, leading to low red-blood cell and low white-blood cell counts, can be seen at any time during treatment and generally is reversible with drug discontinuation
- Check patients with less severe disease 2 to 3 weeks after their initial evaluation, and then monthly to bimonthly until medications are tapered and clinical signs are resolved

PREVENTIONS AND AVOIDANCE

- If a food allergy or intolerance is suspected or documented, avoid feeding that particular nutrient and adhere strictly to dietary changes recommended by your pet's veterinarian

POSSIBLE COMPLICATIONS

- Weight loss and debilitation in cases that do not respond or respond poorly to dietary manipulation or medication
- Excessive levels of medication-related steroids in the body lead to signs of hyperadrenocorticism or Cushing's disease; when these signs are caused by medication, the disease is known as "iatrogenic hyperadrenocorticism"
- Steroid side-effects, such as excessive thirst (known as "polydipsia") and excessive urination (known as "polyuria")
- Bone-marrow suppression (leading to low red-blood cell and low white-blood cell counts); inflammation of the pancreas (known as "pancreatitis"); inflammation of the liver (known as "hepatitis"); or lack of appetite (anorexia) caused by azathioprine and/or chlorambucil
- Vomiting, diarrhea, and lack of appetite (anorexia) with cyclosporine; temporarily decreasing the dosage typically will result in resolution of gastrointestinal signs
- "Dry eye" (known as "keratoconjunctivitis sicca" or "KCS") with sulfasalazine

EXPECTED COURSE AND PROGNOSIS

- Dogs and cats with mild-to-moderate inflammation have a good-to-excellent prognosis for full recovery
- Patients with severe disease, particularly if other portions of the gastrointestinal tract are involved, have a more-guarded prognosis
- Often the initial response to therapy sets the tone for a given individual's ability to recover



KEY POINTS

- Inflammatory bowel disease (IBD) is more likely to be controlled, rather than cured, as relapses are common
- Patience is required during the various food and medication trials that often are necessary

GASTROESOPHAGEAL REFLUX



BASICS

OVERVIEW

- “Gastro-” refers to the stomach; “esophageal” refers to the esophagus; the “esophagus” is the tube running from the throat to the stomach
- “Reflux” is the medical term for backward flow
- “Gastroesophageal reflux” is backward or reverse flow of stomach or intestinal contents into the esophagus
- Incidence unknown; probably more common than clinically recognized
- Transient relaxation of the muscle between the stomach and esophagus (known as the “gastroesophageal sphincter”) or long-term (chronic) vomiting may permit backward or reverse flow (reflux) of gastrointestinal juices into the esophagus
- Inflammation of the esophagus (known as “esophagitis”) resulting from reflux may vary from mild inflammation of the superficial lining of the esophagus to severe ulceration involving the deeper layers of the esophagus

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- May be associated with congenital (present at birth) hiatal hernia seen in Chinese shar peis; “hiatal hernia” is a condition in which part of the stomach slips from the abdomen into the chest through the normal opening for the esophagus as it passes through the diaphragm (the opening through the diaphragm is the “esophageal hiatus”)

Mean Age and Range

- Occurs at any age; younger animals may be at increased risk because of developmental immaturity of the muscle between the stomach and esophagus (gastroesophageal sphincter)
- Young animals with congenital (present at birth) hiatal hernia may be at increased risk of developing gastroesophageal reflux; “hiatal hernia” is a condition in which part of the stomach slips from the abdomen into the chest through the normal opening for the esophagus as it passes through the diaphragm (the opening through the diaphragm is the “esophageal hiatus”)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Return of food or other contents from the esophagus or stomach back up through the mouth (known as “regurgitation”)
- Excessive salivation (known as “hypersalivation”)
- Howling or crying during swallowing
- Lack of appetite (known as “anorexia”)
- Weight loss
- Physical examination often is unremarkable
- Fever and excessive salivation (hypersalivation)—with severe ulcerative inflammation of the esophagus (esophagitis)

CAUSES AND RISK FACTORS

- Anesthesia
- Failure to fast an animal prior to anesthesia
- Poor patient positioning during anesthesia
- Hiatal hernia (condition in which part of the stomach slips from the abdomen into the chest through the normal opening for the esophagus as it passes through the diaphragm [the opening through the diaphragm is the “esophageal hiatus”])
- Long-term (chronic) vomiting
- Young age



TREATMENT

HEALTH CARE

- Generally, managed as an outpatient

ACTIVITY

- Not necessary to restrict activity

DIET

- Moderate to severe cases—may withhold food for 1 to 2 days; thereafter, feed low-fat, low-protein meals in small, frequent feedings
- Dietary fat decreases tone of the muscle between the stomach and esophagus (gastroesophageal sphincter) and delays stomach emptying; protein stimulates stomach-acid secretion
- Avoid feeding high-fat foods; they might worsen the backward or reverse flow of stomach or intestinal contents into the esophagus (esophageal reflux)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Oral sucralfate suspension; sucralfate is a medication that forms a protective barrier over ulcers in the gastrointestinal tract
- Medications to decrease the secretion of stomach acid—cimetidine; ranitidine; famotidine; omeprazole
- Drugs that improve the propulsion of contents through the stomach and intestines (known as “gastrointestinal prokinetic agents,” such as cisapride, ranitidine, low-dose erythromycin)—may increase tone of the muscle between the stomach and esophagus (gastroesophageal sphincter)



FOLLOW-UP CARE

PATIENT MONITORING

- Patients do not necessarily require follow-up procedure using a special lighted instrument called an “endoscope” that is passed into the esophagus and stomach through the mouth to allow the veterinarian to see the lining of the esophagus and stomach (general term for procedure is “endoscopy”); however, endoscopy may be considered for patients that do not respond to medical treatment
- Monitor clinical signs

PREVENTIONS AND AVOIDANCE

- Avoid feeding high-fat foods; they might worsen the backward or reverse flow of stomach or intestinal contents into the esophagus (esophageal reflux)

POSSIBLE COMPLICATIONS

- Inflammation of the esophagus (esophagitis)
- Abnormal narrowing of the esophagus (known as “esophageal stricture”)



KEY POINTS

- Avoid feeding high-fat foods; they might worsen the backward or reverse flow of stomach or intestinal contents into the esophagus (esophageal reflux)

GINGIVITIS (INFLAMMATION OF THE GUMS)



BASICS

OVERVIEW

- Reversible inflammation of the gums (gingivitis); earliest phase of periodontal disease (that is, disease of the tissues around and supporting the tooth)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Over 80% of pets 3-years-old and older have inflammation of the gums (gingivitis)
- Toy-breed dogs affected earlier in life
- Cats generally are affected later in life than are dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Swelling of the gum tissue (known as “gingival tissue”)
- Bad breath (known as “halitosis”)
- Redness or fluid build-up (edema) of the gums
- Variable amounts of plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva) and tartar or calculus (mineralized plaque on the tooth surface)
- Gum surfaces bleed easily on contact (for example, during play or physical examination)

CAUSES

- Accumulation of the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva (plaque)

RISK FACTORS

- Age
- Misalignment and crowding of teeth reduce natural cleaning mechanisms (seen in toy-breeds; in short-nosed, flat-faced breeds [known as “brachycephalic breeds”]; and others)
- Toy-breed dogs affected earlier in life
- Soft foods
- Open-mouth breathing
- Chewing habits
- Lack of oral health care
- Metabolic diseases (such as uremia [condition in which excess levels of urea and other nitrogenous waste products are present in the blood] and diabetes mellitus [“sugar diabetes”]) may allow more disease-causing bacteria to grow in the gum tissue
- Autoimmune disease



TREATMENT

HEALTH CARE

- Proper dental cleaning—complete oral examination; removal of plaque and calculus from the surfaces of the teeth above the gum line (known as “supragingival” surfaces); scaling and root planing (if needed) the surfaces of the teeth below the gum line (known as “subgingival” surfaces); polishing; flushing the tissues below the gum line (known as “subgingival irrigation”) with an antiseptic solution; postcleaning examination; home-care instructions; and follow-up examinations
- Home dental care (such as toothbrushing, use of specialized dental diets or treats, application of gels or rinses) is important to control accumulation of the thin, “sticky” film that builds up on the teeth (plaque); your pet’s veterinarian will recommend a home-care dental

plan for your pet and will provide instructions on using the various home-care dental products

- Rawhide chew strips help to clean the teeth mechanically; they should not be relied upon as the sole method of home care

DIET

- Hard food leaves less lingering material on the teeth than soft food; chewing also helps to clean teeth mechanically; specialized dental diets are available, such as Hill's Prescription Diet® t/d® (Hill's Pet Nutrition, Inc., Topeka, KS), which is formulated to reduce plaque (the thin, "sticky" film that builds up on the teeth) and tartar or calculus (mineralized plaque on the tooth surface) and reduce staining; IAMS® Dental Formula is formulated to reduce tartar or calculus (mineralized plaque on the tooth surface) (IAMS Company Inc, Dayton, OH)

SURGERY

- Extraction of retained deciduous teeth (condition in which both the baby tooth [deciduous tooth] and the permanent or adult tooth are present at the same time) and selective extraction of crowded teeth is necessary to improve the health of the mouth and to allow easier cleaning of tooth surfaces



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Lactoperoxidase- and chlorhexidine-containing chemical agents (known as "dentifrices") are effective in retarding plaque (the thin, "sticky" film that builds up on the teeth)
- Topically applied chlorhexidine, 0.4% stannous fluoride gel, and zinc ascorbate also reduce plaque formation
- Antibiotics generally are not necessary at this stage of disease



FOLLOW-UP CARE

PATIENT MONITORING

- Regular oral re-examinations are necessary, so the veterinarian can determine the proper interval between professional dental treatments and assess the effectiveness of oral home care; professional dental treatment and oral home care can cure gingivitis and help avoid progression to inflammation and infection of the tissues around and supporting the tooth (known as "periodontitis")

PREVENTIONS AND AVOIDANCE

- Modify behavior to avoid chewing hard objects (such as rocks and sticks) and eliminate repetitive trauma, if possible
- Stress the importance of regular professional dental treatment and oral home care; daily or at least twice-weekly toothbrushing is recommended, using an enzymatic toothpaste or zinc-ascorbic acid solution to remove and retard plaque (the thin, "sticky" film that builds up on the teeth); if the owner is unwilling or unable to brush the teeth, but the patient is manageable, the owner might bring the pet to the clinic for brushing
- Eliminate predisposing factors, such as retained deciduous teeth (condition in which both the baby tooth [deciduous tooth] and the permanent or adult tooth are present at the same time) and crowded teeth

POSSIBLE COMPLICATIONS

- Gingivitis begins when bacteria invade the gums; inflammation results in swelling and reddening of the gums, which also become fragile and bleed easily; these lesions are reversible with professional dental treatment and oral home care; if not controlled at this point, the attached gum (gingiva) and tooth-supporting structures (alveolar bone, periodontal ligament, and tooth root cementum) become involved, signifying the transition to periodontitis (inflammation and infection of the tissues around and supporting the tooth)
- Once periodontitis is established, it generally is considered controllable, but not reversible
- Uncontrolled periodontitis invariably leads to tooth loss

EXPECTED COURSE AND PROGNOSIS

- Professional dental treatment, followed by oral home care, completely reverses gingivitis



KEY POINTS

- Stress the importance of regular professional dental treatment and oral home care; daily or at least twice-weekly toothbrushing is recommended, using an enzymatic toothpaste or zinc-ascorbic acid solution to remove and retard plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white blood cells, food particles, and components of saliva)
- Professional dental treatment, followed by oral home care, completely reverses gingivitis

GINGIVOSTOMATITIS AND CAUDAL STOMATITIS

(INFLAMMATION OF THE GUMS AND MOUTH)



BASICS

OVERVIEW

- Excessive inflammation, caused by an immune process, affecting the gums and mouth in cats
- “Gingivostomatitis” is inflammation of the gums and other moist tissues (known as “mucous membranes”) of the mouth
- “Stomatitis” is inflammation of the moist tissues of the mouth
- “Caudal” refers to the back or rear; in this condition, it describes the location in the mouth—the back portion of the mouth

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cat

Breed Predilections

- Purebred cats more likely than other cats—Abyssinian, Persian, Himalayan, Burmese, Siamese, and Somali

SIGNS/OBSERVED CHANGES in the ANIMAL

- Excessive salivation/drooling (known as “ptyalism”)
- Bad breath (known as “halitosis”)
- Difficulty swallowing (known as “dysphagia”)
- Lack of appetite (known as “anorexia”)—prefers soft food
- Weight loss
- Scruffy haircoat
- Reddened, ulcerated lesions with rapidly growing tissue (known as “proliferative tissue”) affecting the gums (known as “gingiva”); the folds of moist tissue extending from the soft palate to the side of the tongue (known as the “glossopalatine arches”); tongue; lips; lining of the cheeks (known as “buccal mucosa”); and/or hard palate
- Inflammation of the gums completely surrounds the tooth
- May extend to the folds of moist tissue extending from the tongue to the wall of the throat or pharynx (known as the “glossopharyngeal arches”), as well as the palate

CAUSES

- Unknown
- Bacterial, viral and immune-mediated causes are suspected
- Feline calici virus
- Decreased immune response (known as “immunosuppression”) from feline leukemia virus (FeLV) or feline immunodeficiency virus (FIV) also can lead to poorly responsive infections; however, most cats affected with gingivostomatitis or caudal stomatitis are negative for FeLV and FIV



TREATMENT

HEALTH CARE

- Initial therapy involves professional teeth cleaning (above and below the gums [gingiva]), as well as strict home care
- Dental X-rays should be taken before and after surgery
- Postoperative application of fluocinonide 0.05% (Lidex® Gel) to the gum margin may help in the healing process

SURGERY

- Biopsy (especially for lesions involving only one side of the mouth) to rule out cancer—primarily squamous cell carcinoma
- Extraction of the teeth behind the canine teeth (that is, the premolar and molar teeth) will result in resolution in 60% of the cases,

without further need of medication

- If patient does not respond to extraction of the teeth behind the canine teeth (that is, the premolar and molar teeth), remove all remaining teeth; when extracting the teeth, pay meticulous attention to removing all tooth substance
- CO₂ laser may be used to decrease inflamed tissue



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medication and other therapies have been used with limited long-term success; lack of permanent response to conventional oral hygiene products, antibiotics, anti-inflammatory drugs, and drugs to decrease the immune response (known as “immunosuppressive drugs”) is typical
- Antibiotics—clindamycin, metronidazole, amoxicillin, ampicillin, enrofloxacin, tetracycline
- Steroids—to decrease the immune response; such as prednisone; methylprednisolone also may help control inflammation
- Gold salts—Solganal® (Schering); used to treat serious immune-mediated diseases
- Chlorambucil, a chemotherapy drug to decrease the immune response
- Bovine lactoferrin is a protein found in cow’s milk that may have antibacterial activity and may have an affect on the immune system; it can be applied to the moist tissues (known as “mucous membranes”) of the mouth
- Interferon is a protein produced by cells of the immune system; has a variety of effects in the body, including fighting viruses and cancer
- Cyclosporine, to decrease the immune response



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor response to treatment; if patient does not respond to extraction of the teeth behind the canine teeth (that is, the premolar and molar teeth), remove all remaining teeth

EXPECTED COURSE AND PROGNOSIS

- Cases with extensive lesions of rapidly growing tissue (proliferative tissue) in the back part of the mouth and throat (pharynx) that respond poorly to treatment warrant a more guarded prognosis



KEY POINTS

- Excessive inflammation, caused by an immune process, affecting the gums and mouth in cats
- “Gingivostomatitis” is inflammation of the gums and other moist tissues (known as “mucous membranes”) of the mouth
- Extraction of the teeth behind the canine teeth (that is, the premolar and molar teeth) will result in resolution in 60% of the cases, without further need of medication
- If patient does not respond to extraction of the teeth behind the canine teeth (that is, the premolar and molar teeth), remove all remaining teeth

GLAUCOMA



BASICS

OVERVIEW

- “Glaucoma” is a disease of the eye, in which the pressure within the eye is increased (pressure within the eye is known as “intraocular pressure”)
- High intraocular pressure that causes characteristic degenerative changes in the optic nerve and retina with subsequent loss of vision; the “optic nerve” is the nerve that runs from the back of the eye to the brain; the “retina” is the innermost lining layer (located on the back surface) of the eyeball; it contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision
- Diagnosis—intraocular pressure greater than 25 to 30 mmHg in dogs or greater than 31 mmHg in cats, as determined by specialized pressure measurements (such as applanation, rebound, or Schiötz tonometry) with evidence of changes in vision or appearance of the optic nerve or retina
- Glaucoma may be “primary” or “secondary;” “primary” refers to a condition in which the pressure within the eye (intraocular pressure) increases without a preceding eye problem; “secondary” refers to a condition in which intraocular pressure increases as a complication or secondary to an eye disease or injury

GENETICS

- Dogs—abnormality of the structure of the eye that makes development of glaucoma more likely in some dogs is thought to be inherited; mode of inheritance uncertain

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs—primary and secondary glaucoma
- Cats—primary glaucoma is rare; secondary glaucoma seen in patients with signs of long-standing inflammation of the iris and other areas in the front part of the eye (known as “uveitis”) or with movement of the lens out of its normal location (known as “lens luxation”); the “lens” is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye (retina)

Breed Predispositions

- Developmental abnormality of the angle between the iris and the cornea of the eye (known as “goniodysgenesis”)—Arctic circle breeds (such as Norwegian elkhounds, Siberian huskies, Alaskan malamutes, Akitas, Samoyeds); Bouvier des Flanders; basset hounds; chow chows; Chinese shar peis; spaniels (such as American and English cocker spaniels, English and Welsh springer spaniels)
- Narrow filtration angles—spaniels; chow chows; Chinese shar peis; toy breeds (such as poodles, Maltese, and shih tzus)
- Secondary to movement of the lens out of its normal location (lens luxations)—terriers (such as Boston terriers, Cairn terriers, Manchester terriers, Dandie Dinmont terriers, Norfolk terriers, Norwich terriers, Scottish terriers, Sealyham terriers, West Highland white terriers, Parsons Jack Russell terriers, and fox terriers), Chinese shar peis

Mean Age and Range

- Primary glaucoma in dogs—any age; predominantly affects middle-aged dogs (4 to 9 years of age)
- Secondary to movement of the lens out of its normal location (lens luxations) in dogs—usually affects young dogs (2 to 6 years of age)
- Secondary to long-term (chronic) inflammation of the iris and other areas in the front part of the eye (uveitis) in cats—usually affects older cats (greater than 6 years of age)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden closure of the angle between the iris and cornea of the eye (known as “acute angle closure”), leading to blockage of the flow of fluid and subsequent increased pressure within the eye—apparent pain (squinting or spasmodic blinking [known as “blepharospasm”], tenderness about the head, discharge from the eye(s), may be clear or may contain mucus); may note a cloudy or red eye; vision loss usually not noticed unless both eyes are involved
- Secondary glaucoma—depends on primary disease
- Inflammation of the iris and other areas in the front part of the eye (uveitis)—may note pain (for many days); red or bloodshot eyes, caused by dilated blood vessels (known as “scleral injection”); and cloudiness due to fluid build-up in the clear part of the eye (known as “corneal edema”)
- Movement of the lens out of its normal location and into the front part of the eye (known as “anterior lens luxation”)—may note sudden (acute) pain; red or bloodshot eyes (scleral injection); and cloudiness due to fluid build-up in the clear part of the eye (corneal edema); may see lens in the anterior chamber (the front part of the eye, between the cornea and the iris), if corneal edema is not severe
- Long-term (chronic) inflammation of the iris and other areas in the front part of the eye (uveitis) in cats—may not have signs of

pain; enlarged, seemingly painless eye or a dilated pupil is common

- Eyeball or globe enlargement (known as “buphthalmos”)—may be noticed first by owners

Sudden (Acute) Primary Glaucoma

- High intraocular pressure (measured by your pet’s veterinarian)
- Squinting or spasmodic blinking (blepharospasm)
- Eyeball may recede into back of socket (known as “enophthalmos”)
- Red or bloodshot eyes (scleral injection)
- Cloudiness due to fluid build-up in the clear part of the eye (corneal edema)
- Dilated pupil; the “pupil” is the circular or elliptical opening in the center of the iris of the eye—light passes through the pupil to reach the back part of the eye (known as the “retina”); the “iris” is the colored or pigmented part of the eye—it can be brown, blue, green, or a mixture of colors
- Vision loss
- Optic nerve may be depressed or cupped when the back of the eye is evaluated with specialized eye instruments by your pet’s veterinarian; the “optic nerve” is the nerve that runs from the back of the eye to the brain

Long-Term (Chronic) or End-Stage Glaucoma

- Eyeball or globe enlargement (buphthalmos)
- Lines that develop on the inner lining of the cornea, the normally clear part of the front of the eye (known as “Descemet streaks” or “Haab’s striae”)
- Partial movement of the lens out of its normal location (known as a “lens subluxation”) with a resultant “crescent” appearing in the area of the iris (known as an “aphakic crescent”)
- Wasting away or decrease in size of the cells in the optic nerve head (known as “optic nerve head atrophy”); the “optic nerve” is the nerve that runs from the back of the eye to the brain
- Death of tissue in the retina (known as “retinal necrosis”)—detected increased reflectivity in the back of the eye when the veterinarian performs an examination

Glaucoma Induced by Inflammation of the Iris and Other Areas in the Front Part of the Eye (Uveitis)

- Elevated intraocular pressure
- Red or bloodshot eyes (scleral injection)
- Cloudiness due to fluid build-up in the clear part of the eye (corneal edema)
- Inflammatory debris in the front part of the eye, between the cornea and the iris (anterior chamber)
- Constricted or miotic pupil may or may not be seen; the “pupil” is the circular or elliptical opening in the center of the iris of the eye; light passes through the pupil to reach the back part of the eye (known as the “retina”); the “iris” is the colored or pigmented part of the eye—it can be brown, blue, green, or a mixture of colors
- Scar tissue between the iris and the lens of the eye (known as “posterior synechia”) may or may not be present; the “lens” is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye (retina)
- Bulging of the iris toward the front of the eye (known as “iris bombé”) may or may not be present

CAUSES

- Primary glaucoma—structural abnormalities of the eye involving the filtration angle (the “filtration angle” is the area where the cornea, sclera, and iris meet; it contains a structure that allows fluid to flow out of the eye, thus maintaining normal pressure within the eye; in primary glaucoma, the structure is abnormal so fluid does not flow adequately and the pressure within the eye increases)
- Secondary glaucoma—blockage of the flow of aqueous humor out of the eye; the “aqueous humor” is the transparent liquid that fills the front part of the eyeball

RISK FACTORS

- Inflammation of the front part of the eye, including the iris (known as “anterior uveitis”)
- Movement of the lens out of its normal location (lens luxation)
- Blood in the anterior chamber of the eye (the front part of the eye, between the cornea and the iris; accumulation of blood known as “hyphema”)
- Tumor or cancer within the eyeball
- Medications applied to the eye directly to dilate the pupil (known as “mydriatics”)—may lead to sudden (acute) glaucoma in susceptible animals
- Primary glaucoma in dogs—consider all cases to involve both eyes, even if one eye has normal intraocular pressure at the time of evaluation; a veterinary eye doctor (ophthalmologist) should examine the apparently normal eye for filtration angle abnormalities to determine the risk for future glaucoma



TREATMENT

HEALTH CARE

- Sudden (acute) glaucoma in dogs—inpatient care
- After discharge from the veterinary hospital—re-evaluate every 1 to 2 days for 1 week to monitor for return of increased intraocular pressure

SURGERY

- Cases with primary glaucoma or glaucoma due to movement of the lens out of its normal location (lens luxation) induced are best treated surgically
- Primary glaucoma in dogs—less than 10% of patients undergoing medical treatment alone will have vision remaining at the end of the first year following diagnosis
- Various surgical procedures may be performed to increase the flow of aqueous humor out of the eye; to decrease production of aqueous humor in the eye; procedures performed attempt to maintain normal intraocular pressure and vision; the “aqueous humor” is the transparent liquid that fills the front part of the eyeball
- Surgical removal of lens that has moved forward in the eye (anterior lens luxation) may result in a visual eye, as well as help lower intraocular pressure
- Blind, painful eyes—surgically remove the eye (known as “enucleation”); may remove the inner parts of the eye surgically, leaving the eyeball and place a prosthesis in the eye (known as “evisceration and intraocular prosthesis implantation”) in some cases



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Use multiple agents to lower intraocular pressure into the normal range as quickly as possible in an attempt to salvage vision

Sudden (Acute) Primary Glaucoma in Dogs

- Emergency medical treatment may include one or more of the following:
- Medications applied to the eye directly to cause the pupil to constrict (known as “topical miotics”)—latanoprost 0.005% (Xalatan®), travoprost 0.004% (Travatan®), or bimatoprost 0.03% (Lumigan®); 2% pilocarpine solution; 0.25% demecarium bromide; treatment aimed at improving aqueous outflow
- Topical beta-adrenergic antagonists—timolol maleate 0.5%, levobunolol 0.5%, betaxolol 0.5%; reduce aqueous humor production; the “aqueous humor” is the transparent liquid that fills the front part of the eyeball
- Carbonic anhydrase inhibitor (administered by mouth)—methazolamide; reduce production of aqueous humor
- Carbonic anhydrase inhibitors (applied to the eye directly)—dorzolamide 2% (TruSopt®), brinzolamide 1% (Azopt®); reduce aqueous humor production
- Medications to remove fluids from the body (known as “hyperosmotic agents”)—mannitol or glycerin; dehydrate the vitreous humor; the “vitreous” is the clear, gel-like material that fills the back part of the eyeball (between the lens and the retina)

Glaucoma Secondary to Movement of the Lens Out of its Normal Location Toward the Front of the Eye (Anterior Lens Luxation) or Inflammation of the Iris and Other Areas in the Front Part of the Eye (Uveitis) in Dogs

- Treated like primary glaucoma
- Medications to cause the pupil to constrict (miotic agents) should not be used
- Steroids applied to the eye directly (known as “topical steroids”)—used to reduce inflammation if no changes of the cornea (the clear outer layer of the front of the eye) characterized by the presence of ulcers, with or without inflammation (condition known as “ulcerative keratitis”)

Long-Term (Chronic) Smoldering Inflammation of the Iris and Other Areas in the Front Part of the Eye (Uveitis) in Cats

- Steroids applied to the eye directly (topical steroids)
- Topical beta-blockers; reduce aqueous humor production; the “aqueous humor” is the transparent liquid that fills the front part of the eyeball
- Carbonic anhydrase inhibitor diuretics and topical carbonic anhydrase inhibitors to reduce production of aqueous humor



FOLLOW-UP CARE

PATIENT MONITORING

- Intraocular pressure—monitored often and regularly after starting initial therapy; if low intraocular pressure (known as “hypotensive ocular pressure”) is maintained for many weeks, *slowly* taper drug therapy (as directed by your pet’s veterinarian)
- Monitor for drug reactions

PREVENTIONS AND AVOIDANCE

- Primary glaucoma—consider all cases to involve both eyes, even if one eye has normal intraocular pressure at the time of evaluation; a veterinary eye doctor (ophthalmologist) should examine the apparently normal eye for filtration angle abnormalities to determine the risk for future glaucoma
- Prophylactic therapy for the apparently unaffected eye—0.25% demecarium bromide or 0.005% latanoprost or 0.5% timolol maleate or 2% dorzolamide; delays onset of glaucoma in second susceptible eye

POSSIBLE COMPLICATIONS

- Blindness
- Long-term (chronic) eye pain

EXPECTED COURSE AND PROGNOSIS

- Long-term (chronic) disease that requires constant medical treatment (even with surgical intervention)
- With medical treatment only—most patients ultimately go blind
- Surgical treatment—better chance of retaining vision longer; most patients do not remain visual for more than 2 years after initial diagnosis
- Secondary glaucoma due to movement of the lens out of its normal location (lens luxation)—may carry a fair prognosis with successful surgical removal of the luxated lens
- Secondary glaucoma due to anterior uveitis—may carry a fair prognosis with control of inflammation of the iris and other areas in the front part of the eye



KEY POINTS

- Primary glaucoma is a disease that involves both eyes; over 50% of animals develop glaucoma in the other eye within 8 months without prophylactic therapy
- 40% or more of dogs will be blind in the affected eye within the first year, no matter what is done medically or surgically

GLOMERULONEPHRITIS

(KIDNEY INFLAMMATION INVOLVING THE GLOMERULUS, THE “BLOOD FILTER”)



BASICS

OVERVIEW

- The kidney filters the blood and removes various waste products from the body as it produces urine; the kidney also is involved in maintaining the normal fluid volume of the body; each kidney is composed of thousands of nephrons (the functional units of the kidney, each consisting of the glomerulus [a tuft of blood capillaries—the “blood filter”] and a series of tubes and ducts, through which the filtered fluid flows, as urine is produced)
- Glomerulonephritis is inflammation and accompanying dysfunction of glomeruli (plural of glomerulus); most commonly due to the presence of immune complexes in the glomerulus
- In veterinary medicine, “glomerulonephritis” has been used as an “umbrella” term for all glomerular diseases; however, certain glomerular diseases (membranous glomerulopathy; kidney amyloidosis [condition in which insoluble proteins, amyloid, are deposited outside the cells in the kidney, compromising its normal function]; and most inherited kidney diseases) are not truly types of “glomerulonephritis,” as the primary lesion is not due to inflammation of the glomerulus

GENETICS

- Familial glomerular disease (glomerular disease that runs in certain families of animals) has been reported in Bernese mountain dogs, bull terriers, Dalmatians, Samoyeds, Doberman pinschers, cocker spaniels, Newfoundlands, rottweilers, greyhounds, soft-coated wheaten terriers, and cats
- Familial amyloidosis (condition that runs in certain families of animals, in which insoluble proteins [amyloid] are deposited outside the cells in the kidney, compromising its normal function) is common in Chinese shar peis; sporadic cases of affected litters have been reported in other breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs; less commonly, cats

Breed Predislection

- See **GENETICS**
- In some studies, golden retrievers, miniature schnauzers, and long-haired dachshunds appear to be more likely to develop glomerulonephritis than other breeds (with exception of breeds listed in **GENETICS**); however, these findings may reflect breed popularity rather than a true increase in likelihood of disease
- Labrador and golden retrievers appear to be more likely to develop glomerulonephritis; sudden (acute) death of cells and tissue of the kidney tubules (known as “tubular necrosis”); and interstitial inflammation associated with Lyme disease (*Borrelia burgdorferi* infection)

Mean Age and Range

- Dogs—mean age, 6.5 to 8.5 years; range, 0.8 to 17 years; dogs with kidney inflammation due to a genetic abnormality (known as “hereditary nephritis”) usually have changes in the kidney at very young ages, prior to the onset of seeing protein in the urine (known as “proteinuria”)
- Cats—mean age at presentation, 4 years

Predominant Sex

- Dogs—no difference between males and females
- Cats—75% are males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs depend on severity and duration of proteinuria (protein in the urine) and kidney failure
- Significant proteinuria (protein in the urine) often is discovered on yearly health screens or while evaluating other problems
- Occasionally, signs associated with an underlying infection, inflammation, or cancer may be the reasons why owners seek veterinary care
- If protein loss into the urine is mild-to-moderate, dogs usually are asymptomatic (that is, show no signs of kidney disease); however, nonspecific signs may include weight loss and sluggishness (lethargy)
- If protein loss into the urine is severe, (in which levels of albumin [a type of protein] in the blood drop to less than 1.5 g/dL), fluid build-up (known as “edema”) in body tissues and/or fluid build-up in the abdomen (known as “ascites”) often occurs; albumin normally plays a major role in holding fluid within the blood/circulation—when the levels of albumin drop to less than 1.5 g/dL, fluids move from the circulation into surrounding tissues, leading to edema or ascites
- If the disease has progressed to kidney failure, excessive urination (known as “polyuria”), excessive thirst (known as “polydipsia”),

lack of appetite (known as “anorexia”), nausea, and vomiting may occur

- Sudden (acute) difficulty breathing (known as “dyspnea”) or severe panting in dogs may be caused by blood clots in the lungs (known as “pulmonary thromboembolism”), an uncommon development, which occurs in association with moderate-to-severe low levels of albumin in the blood (known as “hypoalbuminemia,” with serum albumin concentration of less than 2.5 g/dL)
- Sudden (acute) blindness may occur due to bleeding in the retina or back part of the eye (known as “retinal hemorrhage”) or to detachment of the retina; may be associated with systemic high blood pressure (hypertension)

CAUSES

Several infectious and inflammatory diseases have been associated with deposits or formation of immune complexes in the glomerulus. In many cases, no antigen source or underlying disease process is identified, so the glomerulonephritis is considered to be “idiopathic” (meaning that no cause is known). The following diseases have been associated with glomerulonephritis:

- Dogs—infectious disease (such as, infectious canine hepatitis [viral inflammation of the liver]; bacterial endocarditis [bacterial infection/inflammation of the lining of the heart]; brucellosis [infection caused by *Brucella canis*]; dirofilariasis [heartworm infection]; ehrlichiosis [infection caused by *Ehrlichia*]; leishmaniasis [infection caused by *Leishmania*]; pyometra [inflammation/infection of the uterus]; borreliosis [infection caused by *Borrelia burgdorferi*, also known as “Lyme disease”]; any chronic bacterial infection); cancer; inflammatory diseases; endocrine or hormonal diseases (such as excessive production of steroids by the adrenal glands [known as “hyperadrenocorticism” or “Cushing’s disease”]; diabetes mellitus; long-term administration of steroids); inherited kidney disorders; miscellaneous causes (such as medications; for example, sulfonamides)
- Cats—infectious disease (such as feline leukemia virus [FeLV] infection; feline infectious peritonitis [FIP]; feline immunodeficiency virus [FIV] infection; and *Mycoplasma*-caused inflammation of several joints [known as “polyarthritis”]); cancer; familial (condition that runs in certain families of cat)
- True “autoimmune” glomerulonephritis in which antibodies are directed against the kidney has been documented in only one dog and has not been documented in cats



TREATMENT

HEALTH CARE

- Most patients can be treated as outpatients; exceptions include patients that have very high levels of urea and other nitrogenous waste products in the blood (condition known as “uremia” or “azotemia”) and/or high blood pressure (hypertension); patients with blood-clotting disease (known as “thromboembolic disease”); and patients with fluid build-up in the space between their lungs and chest wall (known as “pleural effusion”) or fluid-build up in the lungs (known as “pulmonary edema”) secondary to low levels of albumin (a protein) in the blood (known as “hypoalbuminemia”)
- Since most glomerular diseases are caused by immune mechanisms, the most specific and effective therapy is elimination of the source of antigenic stimulation (that is, the substance to which the immune system is responding and producing antibodies); often this is difficult, because the disease process or antigen source is not identified or is impossible to eliminate (such as cancer)

ACTIVITY

- Restrictions usually are not necessary
- Patients with severely low levels of albumin in the blood (hypoalbuminemia) may benefit from exercise restriction, because of the possibility of developing blood clots (thromboembolic disease)

DIET

- Sodium-reduced, high-quality, low-quantity protein diets; many manufactured “renal or kidney diets” meet these criteria



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Angiotensin-converting enzyme (ACE) inhibitors, specifically enalapril, decrease loss of protein into the urine (proteinuria); the decrease in protein loss may slow the progression of kidney disease in dogs with idiopathic glomerulonephritis (glomerulonephritis of unknown cause), because protein itself is directly toxic to kidney tubules
- ACE-inhibitor therapy should be initiated at the time of diagnosis, unless severely high levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) are present
- Although glomerulonephritis has an immune basis, no controlled clinical trials in veterinary medicine have demonstrated any benefit

from drugs designed to suppress the immune response (known as “immunosuppressive therapy”); on the contrary, steroids and cyclosporine (immunosuppressive drugs) have been shown to worsen prognosis in many patients

- Aspirin decreases production of thromboxane, a major cause of glomerular inflammation, and decreases platelet clumping and resultant blood-clotting disease (known as “thromboembolic disease”); low-dose aspirin therapy usually is initiated once serum albumin is below 2.5 g/dL
- Use of ACE-inhibitors (other than enalapril) has not been studied in dogs with naturally-occurring glomerular disease



FOLLOW-UP CARE

PATIENT MONITORING

- Follow the urine protein:creatinine (UP/C) ratio closely to determine progression or remission of glomerular disease (known as “glomerulopathy”)
- Magnitude of protein loss into the urine (proteinuria) will decrease as more nephrons (the functional units of the kidney) are lost to progressive disease; therefore, always interpret changes in the urine protein: creatinine (UP/C) ratio in light of changes in serum creatinine concentration and urine specific gravity (measurement of the dissolved substances in a solution, in this case urine; it is used to evaluate the ability of the kidney tubules to remove water from [that is, concentrate] or add water to [that is, dilute] the urine)
- Monitor blood work (such as serum urea nitrogen, creatinine, albumin, and electrolyte concentrations), blood pressure, and body weight
- Ideally, reexamine 1, 3, 6, 9, and 12 months after initiation of treatment

PREVENTIONS AND AVOIDANCE

- Do not use affected animals of breeds with suspected familial glomerular disease for breeding purposes
- Spay or neuter affected animals of breeds with suspected familial glomerular disease

POSSIBLE COMPLICATIONS

- Nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin)
- High blood pressure (hypertension)
- Long-term (chronic) kidney failure
- Blood-clotting disorders (thromboembolic disorders)

EXPECTED COURSE AND PROGNOSIS

- Long-term prognosis is guarded to poor
- Often progresses to chronic kidney failure, despite treatment



KEY POINTS

- If the underlying cause cannot be identified and corrected, the disease often is progressive, resulting in chronic kidney failure
- Once azotemia (high levels of urea and other nitrogenous waste products in the blood; also known as “uremia”) and kidney failure develop, prognosis often is poor due to rapidly progressive disease

STERILE NODULAR/GRANULOMATOUS SKIN DISEASES



BASICS

OVERVIEW

- Skin diseases in which the primary lesions are nodules that are solid, elevated, and greater than 1 cm in diameter

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Nodular dermatofibrosis—German shepherd dogs
- Calcinosis circumscripta—German shepherd dogs
- Malignant histiocytosis—Bernese mountain dogs

Mean Age and Range

- Nodular dermatofibrosis—German shepherd dogs, 3 to 5 years of age
- Calcinosis circumscripta—German shepherd dogs, less than 2 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on cause
- Hair loss (known as “alopecia”)
- May see lesions over pressure points (such as skin over bones)
- Nodules may be found anywhere on body; usually solid and elevated above the surface of the skin
- Lesions containing calcium may be very firm to hard
- Lesions may be tender or painful to the animal

CAUSES

- Amyloidosis
- Foreign body reaction
- Spherulocytosis
- Idiopathic sterile granuloma and pyogranuloma
- Canine eosinophilic granuloma
- Calcinosis cutis
- Calcinosis circumscripta
- Malignant histiocytosis
- Cutaneous histiocytosis
- Sterile panniculitis
- Nodular dermatofibrosis
- Cutaneous xanthoma

RISK FACTORS

- Foreign body reaction—induced by exposure to any irritating material (such as concrete dust or fiberglass)
- Hair foreign bodies—increased risk for large dogs that rest on very hard surfaces
- Calcinosis cutis—increased risk with exposure to high doses of steroids
- Panniculitis—increased risk with vitamin E–deficient diet



TREATMENT

HEALTH CARE

- Most of these disorders can be treated on an outpatient basis
- Dogs with calcinosis cutis may need to be hospitalized for generalized bacterial infection (known as “sepsis”) and intense topical therapy

SURGERY

- Amyloidosis—if the lesion is solitary, it may be removed surgically
- Spherulocytosis—only effective treatment is surgical removal
- Foreign body reactions—best treated by removal of the offending substance, if possible

- Calcinosis circumscripta—surgical removal is the treatment of choice in most cases
- Sterile panniculitis—single lesions can be removed surgically



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Amyloidosis—no known therapy (unless the lesion is solitary and can be removed surgically)
- Idiopathic sterile granuloma and pyogranuloma—prednisone is the first line of therapy; continue steroids for 7 to 14 days after complete remission; then taper dose; for cases that are not responsive to steroids, azathioprine in combination with prednisone or sodium iodide may be tried
- Foreign body reactions—best treated by removal of the offending substance, if possible; for hair foreign bodies, the dog should be placed on softer bedding and topical therapy with agents to soften and loosen crusts and scales on the skin (known as “keratolytic agents”) should be initiated; many dogs with hair foreign bodies also have secondary deep bacterial infections that need to be treated with both topical (applied directly to the skin lesions) and systemic (administered by injection or mouth) antibiotics
- Canine eosinophilic granuloma—prednisone generally produces a good response
- Cutaneous histiocytosis—high-dose steroids and chemotherapeutic drugs result in remission; recurrences are common; l-asparaginase has been helpful in some cases
- Calcinosis cutis—underlying disease must be controlled, if possible; most cases require antibiotics to control secondary bacterial infections; hydrotherapy (which may be achieved with either a whirlpool bath or by spraying cool water under pressure against the affected skin) and frequent bathing in antibacterial shampoos minimize secondary problems; topical DMSO is useful (applied to no more than one-third of the body once daily until lesions resolve)
- Sterile panniculitis—prednisone is the treatment of choice; administered until lesions regress; then tapered; some dogs remain in long-term remission, but others require prolonged alternate-day therapy; a few cases respond to oral vitamin E
- Nodular dermatofibrosis—skin condition seen in German shepherd dogs; no therapy for most cases, because these dogs usually also have kidney disease or cancer; if cancer involves only one kidney, surgical removal of the affected kidney may be helpful.
- Cutaneous xanthoma—correction of the underlying diabetes mellitus (sugar diabetes) or correction of the increased lipoprotein content of the blood (lipoproteins are complexes of lipids [compounds that contain fats or oils] and protein; condition known as “hyperlipoproteinemia”) is usually curative



FOLLOW-UP CARE

PATIENT MONITORING

- Patients on long-term steroids should have a complete blood count (CBC), blood chemistry screen, urinalysis, and urine culture done every 6 months
- Dogs being treated with DMSO for calcinosis cutis should have blood calcium levels checked every 7 to 14 days, starting at the beginning of treatment

POSSIBLE COMPLICATIONS

- Death from generalized (systemic) amyloidosis, malignant histiocytosis, and nodular dermatofibrosis

EXPECTED COURSE AND PROGNOSIS

- Depend on cause
- Generalized (systemic) amyloidosis, malignant histiocytosis, and nodular dermatofibrosis—invariably fatal
- Malignant histiocytosis—no effective therapy; it is rapidly fatal



KEY POINTS

- Skin diseases in which the primary lesions are nodules that are solid, elevated, and greater than 1 cm in diameter

BAD BREATH (HALITOSIS)



BASICS

OVERVIEW

- Offensive odor coming from the mouth; bad breath (halitosis)

SIGNALMENT/DESCRIPTION of ANIMAL

- Dog and cat
- Small breeds and short-nosed, flat-faced breeds (known as “brachycephalic breeds”) are more prone to disease involving the mouth, because their teeth are closer together
- Older animals are more likely to have bad breath (halitosis) than are young animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Bad breath or halitosis is a sign itself
- If due to oral disease, excessive salivation (known as “ptyalism”), with or without blood, may be seen; the animal may paw at the mouth; and lack of appetite (anorexia) may occur
- In most cases, no clinical signs other than actual odor are observed

CAUSES

- Metabolic—diabetes mellitus (“sugar diabetes”), uremia (excess levels of urea and other nitrogenous waste products in the blood)
- Respiratory—inflammation of the nose or nasal passages (known as “rhinitis”); inflammation of the sinuses (known as “sinusitis”); cancer
- Gastrointestinal—enlargement of the esophagus (the tube going from the throat to the stomach; condition known as “megaesophagus”); cancer; foreign body
- Dermatologic—infection of the skin folds of the lips (known as “lip-fold pyoderma”)
- Dietary—eating malodorous or offensive-smelling foodstuffs; eating feces or bowel movement (known as “coprophagy”)
- Disease of the mouth—infection of the gums and supporting tissues of the teeth (known as “periodontal disease”) and/or ulceration of the tissues of the mouth; inflammation of the throat or pharynx (known as “pharyngitis”); inflammation of the tonsils (known as “tonsillitis”); cancer; foreign bodies
- Trauma—electric-cord injury, open fractures, caustic agents
- Infectious—bacterial, fungal, viral
- Autoimmune diseases
- Diseases characterized by one or more masses or nodular lesions in the mouth containing a type of white-blood cell, called an eosinophil (known as “eosinophilic granuloma complex”)

RISK FACTORS

- Small breeds and short-nosed, flat-faced breeds (known as “brachycephalic breeds”) are more prone to disease involving the mouth, because their teeth are closer together; smaller animals live longer; and their owners tend to feed softer food



TREATMENT

HEALTH CARE

- Once the specific cause of the bad breath (halitosis) is known, direct therapy at correcting the cause; it is possible that multiple causes may be involved (for example, the animal may have infection of the gums and supporting tissues of the teeth [periodontal disease] and have a foreign body or cancer present in the mouth)
- Dental disease—assessment of the mouth, performed under general anesthesia, with X-rays of the mouth (known as “intraoral radiographs”) and treatment, including cleaning and polishing the teeth and extraction of teeth with greater than 50% loss of supporting tissues (gum and bone) around the teeth
- Cancer of the mouth—surgical debulking (removing as much of the tumor as possible) or removal; radiation therapy; other cancer therapies, based on type of cancer

- Foreign body—removal of foreign body (may require anesthesia)
- Dermatologic causes—treatment for infection of the folds of the lips may include antibiotics, antibacterial shampoos, and possible surgery to remove some of the folded tissue
- Dietary causes—prevent pet from eating malodorous foodstuffs (for example, keep pet away from garbage); prevent pet from eating bowel movement (for example, block off litter box so dog cannot get to cat feces; clean yard frequently)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medication is determined by the underlying cause of the bad breath (halitosis)
- Topical treatment with zinc-ascorbate cysteine gel usually reduces bad breath within 30 minutes of application, because of the effect of cysteine on sulfur compounds in the mouth
- Antibiotics are not indicated to treat bad breath (halitosis) without performing complete assessment and treatment of the mouth; antibiotics are indicated in the treatment of infection of the lip folds and for cases of rhinitis and/or sinusitis, if bacterial infection is involved
- Controlling the bacteria that cause infection of the gums and supporting tissues of the teeth (periodontal disease) helps control dental infections and accompanying bad breath
- Weekly application of OraVet™ (Merial), a plaque prevention gel has been shown to decrease plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva)
- The use of oral home-care products that contain metal ions, especially zinc, inhibits odor formation because of the affinity of the metal (zinc) ion to sulfur; zinc complexes with hydrogen sulfide to form insoluble zinc sulfide, decreasing the odor
- Chlorhexidine used as a rinse or paste also helps control plaque (the thin, “sticky” film that builds up on the teeth), decreasing eventual odor; many dental home-care products containing chlorhexidine are available commercially
- Zinc ascorbate plus amino acid (Maxi/Guard® Oral Cleansing Gel—Addison Biological Laboratory)
- DenTees™ (Dermapet®) neutralize odors and freshens breath



FOLLOW-UP CARE

PATIENT MONITORING

- Periodic examinations to monitor results of dental professional and home care

PREVENTIONS AND AVOIDANCE

- Varies with underlying cause
- Daily brushing to remove plaque (the thin, “sticky” film that builds up on the teeth) and control dental disease and odor
- Prevent pet from eating malodorous foodstuffs (for example, keep pet away from garbage); prevent pet from eating bowel movement (for example, block off litter box so dog cannot get to cat feces; clean yard frequently)

POSSIBLE COMPLICATIONS

- Varies with underlying cause

EXPECTED COURSE AND PROGNOSIS

- Varies with underlying cause



KEY POINTS

- Bad breath or halitosis is a sign; it is an offensive odor coming from the mouth
- Once the specific cause of the bad breath (halitosis) is known, direct therapy at correcting the underlying cause

HEAD TILT



BASICS

OVERVIEW

- Tilting of the head to one side, away from its normal orientation with the body and limbs
- Usually associated with disorders of the vestibular system, which maintains the animal's normal sense of balance

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Tilting of the head to one side
- Short, rapid movements of the eyeball (known as “nystagmus”)
- Drooling, nausea, vomiting
- “Drunken” or incoordinated gait (known as “ataxia”)
- Disorientation
- Rolling
- Other signs depend on underlying cause of the head tilt

CAUSES

Peripheral Nervous System Disease

- Anatomic abnormality—congenital (present at birth) head tilt
- Metabolic—inadequate levels of thyroid hormone (known as “hypothyroidism”); pituitary gland tumor (pituitary chromophobe adenoma); disease related to the presence of cancer somewhere in the body (known as “paraneoplastic disease”)
- Tumors or cancer—nerve sheath tumor of the vestibulocochlear nerve (also known as “cranial nerve VIII;” the cranial nerves are the nerves that originate in the brain); cancer of the bone and surrounding tissue (types of cancer include osteosarcoma, fibrosarcoma, chondrosarcoma, and squamous cell carcinoma)
- Inflammatory disease—infection/inflammation of the middle and inner ear (known as “otitis media” and “otitis interna”); primarily bacterial infection but also parasitic (such as ear mites, *Otodectes*), mycotic or fungal infections; foreign body; inflammatory masses that develop from the middle ear or eustachian tube (known as “nasopharyngeal polyps”) in cats
- Unknown cause (so called “idiopathic disease”)—canine geriatric vestibular disease; feline idiopathic vestibular disease
- Immune-mediated disease—disorder of the cranial nerve (known as “cranial nerve neuropathy”); the cranial nerves are the nerves that originate in the brain
- Toxic—aminoglycosides, lead, hexachlorophene
- Trauma—fracture of bones near the ear; ear flush

Central Nervous System Disease

- Disease leading to loss of function (known as “degenerative disease”) of nervous tissue—inherited metabolic diseases in which harmful levels of materials accumulate in the body's cells and tissues (known as “storage diseases”); disease in which myelin (a white material that covers certain nerve fibers) is lost (known as “demyelinating disease”); loss of nervous tissue due to a disruption in blood flow to the area (known as a “vascular event”)
- Anatomic abnormality—build-up of fluid in specific areas of the brain (known as “hydrocephalus” or “water on the brain”)
- Tumor or cancer—types of tumors or cancer include glioma, choroid plexus papilloma, meningioma, lymphoma, nerve-sheath tumor, medulloblastoma, skull tumor (such as osteosarcoma); spread of cancer (known as “metastasis,” such as spread of hemangiosarcoma or melanoma into the central nervous system)
- Nutritional disorders—thiamine (a B vitamin) deficiency
- Inflammatory, infectious disease—viral (such as feline infectious peritonitis [FIP], canine distemper); protozoal (such as toxoplasmosis, neosporosis); fungal or mycotic (such as cryptococcosis, blastomycosis, histoplasmosis, coccidioidomycosis, nocardiosis); bacterial (such as extension into the central nervous system from infection/inflammation of the middle and inner ear [otitis media and interna]); parasitic (such as migration of *Cuterebra* larvae); rickettsial (such as ehrlichiosis); algae (protothecosis)
- Inflammatory, noninfectious—inflammation of the brain and spinal cord and the membranes covering them (known as “meninges”) characterized by nodular, inflammatory lesions (known as “granulomatous meningoencephalomyelitis”), breed-specific inflammation of the brain and the membranes covering the brain (known as “meningoencephalitis,” such as Yorkshire terrier necrotizing encephalitis—

inflammation of the brain characterized by the destruction and death of nervous tissue)

- Trauma—fracture of the bone near the ear, with brainstem injury
- Toxic—metronidazole

RISK FACTORS

- Inadequate levels of thyroid hormone (hypothyroidism)
- Administration of medications or products that are toxic to the ear (known as “ototoxic drugs”)
- Thiamine (vitamin B)-deficient diet (such as seen when cat is fed exclusively fish diet)
- Infection/inflammation of the outer ear, middle ear, and inner ear (otitis externa, media, and interna)



TREATMENT

HEALTH CARE

- Inpatient versus outpatient—depends on severity of the signs (especially severity of “drunken” or incoordinated gait [vestibular ataxia]), size and age of the patient, and need for supportive care
- Supportive fluids—replacement or maintenance fluids (depend on clinical state); may be required in the sudden (acute) phase when disorientation, nausea, and vomiting prevent the animal from drinking and eating; especially important in senior patients

ACTIVITY

- Restrict activity (such as avoiding stairs and slippery surfaces) to protect the animal, according to the degree of loss of equilibrium; “equilibrium” is the state or condition of being balanced

DIET

- Usually no need for modification of the diet, unless the cause of the head tilt is thiamine deficiency (such as a cat being fed exclusively fish diet without vitamin supplementation)
- Intake of food and/or water may need to be restricted in cases with nausea and vomiting
- Feed carefully, animals with severe head tilt and vestibular lack of equilibrium or brainstem dysfunction may be prone to aspiration secondary to abnormal body posture

SURGERY

- Surgery may be indicated in some cases of inflammation of the middle ear (otitis media) to drain the middle ear (procedure known as “bullae osteotomy”)
- Surgery may be necessary to remove inflammatory masses that develop from the middle ear or eustachian tube (nasopharyngeal polyps) in cats and to remove tumors (if they are accessible surgically)
- Specific fracture repair or removal of accumulated blood (known as a “hematoma”) is difficult
- Discuss the risks associated with biopsy, surgery, and radiation of a brainstem mass with your pet’s veterinarian



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Infection/inflammation of the middle ear and inner ear (otitis media and interna)—broad-spectrum antibiotic that penetrates bone, while awaiting bacterial culture and sensitivity results; examples include trimethoprim-sulfa; first-generation cephalosporins, such as cephalexin; or amoxicillin/clavulanic acid; treatment often required for 4 to 6 weeks
- Inadequate levels of thyroid hormone (hypothyroidism)—thyroid hormone (T_4) replacement, such as levothyroxine in dogs; response varies, partly depending on the duration of signs (in some patients, the nervous system disorder [neuropathy] is not reversible)
- Medication adversely affecting vestibular function—discontinue use of the medication, as directed by your pet’s veterinarian; signs are usually, but not always, reversible
- Infectious disease—specific treatment, if indicated; for bacterial diseases, antibiotic that penetrates the blood–brain barrier (such as trimethoprim-sulfa or metronidazole); for protozoal diseases, clindamycin; for fungal or mycotic diseases, itraconazole
- Inflammation of the brain and spinal cord and the membranes covering them (meninges) characterized by nodular, inflammatory lesions (granulomatous meningoencephalomyelitis)—usually initially treated with steroids: dexamethasone followed by prednisone;

depending on progress, may need stronger medications to decrease the immune response (known as “immunosuppression”)—azathioprine; cytosine arabinoside; or radiation therapy

- Trauma—supportive care (such as anti-inflammatory drugs, antibiotics, intravenous fluids)
- Canine geriatric and feline idiopathic vestibular disease—usually supportive care only
- Disorder involving several cranial nerves (cranial nerve polyneuropathy)—response to prednisone usually good, if the patient has a primary immune-mediated disorder
- Thiamine deficiency—diet modification and thiamine (a B vitamin) replacement



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat the nervous system examination at a frequency dictated by the underlying cause of the head tilt
- Cases with inadequate levels of thyroid hormone (hypothyroidism)—measure thyroid hormone (T_4) concentration 4 to 6 hours after administration of the thyroid hormone; should be measured at 3 to 4 weeks after initiation of thyroid therapy to evaluate dosage
- Repeat cerebrospinal fluid (CSF) tap and analysis and brain imaging—with some central nervous system vestibular disorders
- Monitor tear production (by doing a Schirmer tear test—technique to measure watery portion of tears) if treating with trimethoprim-sulfa antibiotic
- Monitor complete blood count (CBC), if treating with azathioprine or cytosine arabinoside

POSSIBLE COMPLICATIONS

- Progression of disease with deterioration of mental status
- Brain may push downward in the skull and herniate through the opening that leads to the neck (known as “tentorial herniation” or “brain herniation”), leading to death

EXPECTED COURSE AND PROGNOSIS

- Head tilt may persist
- Prognosis for central nervous system vestibular disorders is usually poorer than that for peripheral nervous system vestibular disorders
- Prognosis usually grave for protozoal, fungal, and viral diseases (such as feline infectious peritonitis [FIP] and canine distemper)



KEY POINTS

- Tilting of the head to one side, away from its normal orientation with the body and limbs
- Usually associated with disorders of the vestibular system, which maintains the animal’s normal sense of balance
- Prognosis for central nervous system vestibular disorders is usually poorer than that for peripheral nervous system vestibular disorders

HEARTWORM DISEASE IN CATS



BASICS

OVERVIEW

- Disease caused by infection with heartworms
- *Dirofilaria immitis* is the scientific name for the heartworm
- Heartworms are spread through the bite of mosquitoes carrying infective heartworm larvae; the life cycle of the heartworm in the dog will be used to help with understanding the disease in cats, since many of the findings in cats are compared to dogs—the heartworm larvae migrate in the dog's body and reach the heart and blood vessels of the lungs in approximately 6 months; adult heartworms grow to about 12 inches long; adult heartworms reproduce and may release immature heartworms (known as “microfilaria”) into the blood of the dog; when a mosquito bites an infected dog, it takes in the microfilaria with its blood meal; the microfilaria undergo development in the mosquito and become infective heartworm larvae, and the life cycle continues
- Presence of immature heartworms (microfilaria) in the blood (known as “microfilaremia”) is uncommon in cats (seen in less than 20% of infected cats)
- Number of cases of heartworm disease in cats is one-tenth that of dogs in a particular geographic location
- Most cats have only a few heartworms present
- Heartworms are physically smaller and have a shorter life span in cats than heartworms in dogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Predominant Sex

- Male cats more commonly infected than are female cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Coughing
- Difficulty breathing (known as “dyspnea”)
- Vomiting
- Blood clots to the lungs (known as “pulmonary thromboembolism”) frequently result in sudden (acute) breathing failure and death
- Vomiting and breathing abnormalities predominate in long-term (chronic) heartworm disease in cats
- Physical examination usually normal
- May hear increased lung sounds when listening to the chest with a stethoscope

CAUSES

- Infection with the heartworm, *Dirofilaria immitis*

RISK FACTORS

- Outdoor cats at increased risk (2:1) of infection
- Feline leukemia virus (FeLV) infection is not a risk factor for heartworm infection in cats



TREATMENT

HEALTH CARE

- Currently no medical treatment to kill the adult heartworms in cats is approved or recommended
- Cats with clinical signs of heartworm disease should be stabilized prior to consideration of worm extraction
- Spontaneous “cure” is probably much more common in cats than dogs (probably due to the shorter heartworm life span in cats as compared to dogs)

SURGERY

- Surgical removal of heartworms or catheter-based extraction of heartworms



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medical treatment to kill the adult heartworms in cats is not approved or recommended

Initial Stabilization

- Supplemental oxygen
- Theophylline (sustained release formulation)
- Prednisolone
- Doxycycline therapy; may reduce severity of lung inflammation secondary to worm death
- Cautiously administer balanced fluids, if indicated

Blood Clots to the Lungs (Pulmonary Thromboembolism)

- Supportive care for blood clots to the lungs (same as initial stabilization)



FOLLOW-UP CARE

PATIENT MONITORING

- Serial evaluation of clinical response
- Chest X-rays
- Heartworm antigen and antibody blood tests are most informative

PREVENTIONS AND AVOIDANCE

- Ivermectin (Heartgard® for Cats)—administered by mouth every 30 days
- Milbemycin oxime (Interceptor®)—administered by mouth every 30 days
- Selamectin (Revolution®)—applied to the skin every 30 days

POSSIBLE COMPLICATIONS

- Blood clots to the lungs (pulmonary thromboembolism)
- Sudden (acute) breathing failure and death



KEY POINTS

- Number of cases of heartworm disease in cats is one-tenth that of dogs in a particular geographic location
- Most cats have only a few heartworms present
- Medical treatment to kill the adult heartworms in cats is not approved or recommended

HEARTWORM DISEASE IN DOGS



BASICS

OVERVIEW

- Disease caused by infestation with heartworms
- *Dirofilaria immitis* is the scientific name for the heartworm
- Heartworms are spread through the bite of mosquitoes carrying infective heartworm larvae; the heartworm larvae migrate in the dog's body and reach the heart and blood vessels of the lungs in approximately 6 months; adult heartworms grow to about 12 inches long; adult heartworms reproduce and may release immature heartworms (known as "microfilaria") into the blood of the dog; when a mosquito bites an infected dog, it takes in the microfilaria with its blood meal; the microfilaria undergo development in the mosquito and become infective heartworm larvae, and the life cycle continues

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilection

- Medium- to large-breed dogs and those living outdoors
- All unprotected dogs are at risk for getting heartworms in areas where heartworm infestation is common (known as "endemic regions")

Mean Age and Range

- Infestation with heartworms can occur at any age; most affected animals are 3 to 8 years of age

Predominant Sex

- Males are affected two-to-four times as often as females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Animals often have no signs or exhibit minimal signs, such as occasional coughing (designated as having "Class I heartworm disease")
- Coughing and exercise intolerance associated with moderate lung damage (designated as having "Class II heartworm disease")
- Extreme weight loss, with muscle wasting (known as "cachexia"); low red-blood cell count (known as "anemia"); exercise intolerance; fainting (known as "syncope"); and fluid build-up in the abdomen (known as "ascites") may be seen in dogs with right-sided congestive heart failure (right-sided CHF) in severely affected dogs (designated as having "Class III heartworm disease"); the "right side" of the heart is the side of the heart that blood enters from the body (blood that has low oxygen levels) and then is pumped into the lungs to get oxygen
- Labored breathing or short, rough snapping sounds (known as "crackles") when listening to the lungs with a stethoscope (known as "auscultation")—dogs with severe elevated blood pressure in the lungs (condition known as "pulmonary hypertension") in Class III heartworm disease or with blood clots in the lungs (known as "pulmonary thromboembolic complications")
- Rapid heart rate (known as "tachycardia"); fluid build-up in the abdomen (ascites); enlargement of the jugular vein, with possible detection of pulses in the jugular vein; and liver enlargement (known as "hepatomegaly") indicate right-sided congestive heart failure (right-sided CHF); may be seen in Class III heartworm disease
- Spitting up of blood derived from the lungs due to pulmonary or bronchial hemorrhage (known as "hemoptysis")—occasionally occurs; indicates severe blood clots in the lungs (pulmonary thromboembolic complications)
- Pale gums and moist tissues of the body (mucous membranes); difficulty breathing (known as "dyspnea"); weak pulses; presence of hemoglobin (the compound in the red-blood cells that carries oxygen to the tissues of the body) in the blood (known as "hemoglobinemia"); and presence of hemoglobin in the urine (known as "hemoglobinuria") are indications of a serious complication of heartworm disease, known as the "caval syndrome"

CAUSES

- Infestation with heartworms (*Dirofilaria immitis*)

RISK FACTORS

- Living in areas where heartworm infestation is common (known endemic regions)
- Most common geographic location is in tropical and subtropical zones
- Heartworm disease is common along the Atlantic/Gulf coasts and Ohio/Mississippi River basins; however, heartworm disease has been diagnosed in dogs in all 50 states
- "Outside" dog increases the risk; but "inside" dogs can be exposed to mosquitoes carrying the infective heartworm larvae
- Lack of heartworm preventive medication or lack of routine use of heartworm preventive medication, as directed by your dog's

veterinarian

- Outside air temperature greater than 64° F all day, every day for at least 1 month (related to mosquito life cycle)
- Outside air temperature greater than 80° F every day for 10 to 14 days (related to mosquito life cycle)



TREATMENT

HEALTH CARE

- Most patients are hospitalized during administration of medication to kill the adult heartworms (known as “adulticide treatment”)
- Eliminate immature heartworms (microfilaria) with monthly heartworm preventive medication, as directed by your dog’s veterinarian; Interceptor® may cause rapid decrease in microfilaria numbers and should be used with caution
- Hospitalization recommended for dogs experiencing blood-clotting problems (thromboembolic complications)

ACTIVITY

- Severe restriction of activity required for 4 to 6 weeks after administration of medication to kill the adult heartworms (adulticide treatment)
- Cage rest and confinement recommended for 3 to 4 weeks after administration of medication to kill the adult heartworms (adulticide treatment) for dogs with severe (Class III) heartworm disease
- Cage rest and confinement for 7 days recommended for dogs experiencing blood-clotting problems (thromboembolic complications)

DIET

- Moderately restricted sodium diet recommended for dogs with signs of congestive heart failure (CHF)

SURGERY

- Treatment of choice for dogs with signs of the serious complication of heartworm disease known as the “caval syndrome”
- Worm removal from the right side of the heart and main artery to the lungs (known as the “pulmonary artery”) via the jugular vein is highly effective for treating high worm burden (that is, a high number of adult worms), when employed by an experienced veterinarian with the appropriate instruments



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Stabilize animals with right-sided congestive heart failure (CHF) with medications to remove excess fluids from the body (known as “diuretics”); angiotensin-converting enzyme (ACE) inhibitors; cage rest; and moderate sodium restriction before administration of medication to kill the adult heartworms (adulticide treatment)
- Stabilize lung/breathing failure with oxygen supplementation; medications to prevent the development of blood clots (known as “antithrombotic agents,” such as aspirin or heparin); or anti-inflammatory dosages of steroids, depending on the clinical and radiographic findings
- Melarsomine (Immiticide®)—a medication to kill the adult heartworms (adulticide); administered by injection; a follow-up positive heartworm-antigen blood test result 4 months later may indicate need for repeat treatment; with a weak positive antigen test result, repeat heartworm-antigen blood test in 1 to 2 months before deciding to repeat adulticide treatment
- Eliminate the immature form of the heartworm (microfilaria) with monthly heartworm preventive medication
- Ivermectin (Heartgard® Plus) administered monthly for at least 32 months kills some adult heartworms (generally not recommended as treatment for heartworm disease)
- Doxycycline has been used by some clinicians prior to administering medications to kill the adult heartworms (adulticide treatment) to eliminate *Wolbachia*, a gram-negative bacteria found in the heartworm that is associated with inflammation of the lungs and kidneys following adulticide treatment



FOLLOW-UP CARE

PATIENT MONITORING

- Perform a heartworm-antigen blood test—4 months after administering medications to kill the adult heartworms (adulticide treatment); if positive, must decide whether or not to repeat the adulticide treatment—if a weak positive test result, repeat the heartworm-antigen blood test in 1 to 2 months
- Some dogs with persistent adult heartworm infestation may not require re-treatment—determined by age, severity of infection, degree of improvement since the first treatment, strength of the positive heartworm-antigen blood test result, and coexistent disease

PREVENTIONS AND AVOIDANCE

- Heartworm preventive medication should be provided for all dogs at risk of getting heartworms
- Heartworm-antigen blood test should be performed prior to starting preventive treatment
- Heartworm-antigen blood test performed 7 months after end of previous mosquito season
- Ivermectin (Heartgard®)—highly effective, monthly heartworm preventive that, when combined with pyrantel pamoate (Heartgard® Plus), also controls hookworm and roundworm infestations; can be given safely to dogs that have immature heartworms (microfilaria) in their blood
- Milbemycin (Interceptor®)—highly effective, monthly heartworm preventive that also controls hookworms, roundworms, and whipworms; the preventive dosage will kill immature heartworms (microfilaria); sudden (acute) reactions may occur when milbemycin is given to dogs that have immature heartworms (microfilaria) in their blood
- Selamectin (Revolution®) is available for monthly topical (applied to the skin) administration as a heartworm preventive
- All of the heartworm preventive medications can be administered safely to collies at the appropriate dosages, as directed by your dog's veterinarian

POSSIBLE COMPLICATIONS

- Blood clots to the lungs following administration of medications to kill the adult heartworms (known as “postadulticide pulmonary thromboembolic complications”)—may occur up to 4 to 6 weeks after treatment; usually more severe in dogs with severe heartworm disease (Class III) and those not properly confined
- Low platelet counts (known as “thrombocytopenia”) and a blood-clotting disorder (known as “disseminated intravascular coagulation” or “DIC”) may occur
- Melarsomine adverse effects—blood clots to the lungs (known as “pulmonary thromboembolism”), usually occur 7 to 30 days after administration of medication to kill the adult heartworms; lack of appetite (known as “anorexia”); injection site reaction with inflammation of the muscle (known as “myositis”)—usually mild and only lasts 1 to 2 days; sluggishness (lethargy) or depression; elevation of liver enzymes on blood tests; partial or complete paralysis or change in mental status (rare)

EXPECTED COURSE AND PROGNOSIS

- Usually uneventful with excellent prognosis in dogs without clinical signs of heartworm disease (asymptomatic dogs) and mildly symptomatic animals (Class I heartworm disease)
- Guarded prognosis with higher risk of complications in dogs with severe (Class III) heartworm disease



KEY POINTS

- Routine heartworm preventive medication (as directed by your dog's veterinarian) is key to preventing your dog from becoming infested with heartworms; heartworm disease is a preventable disease
- Good prognosis for animals with mild-to-moderate heartworm disease
- Complications involving the lungs following administration of medication to kill the adult heartworms (postadulticide pulmonary complications) likely in patients with moderate-to-severe heartworm disease
- Reinfestation can occur, unless appropriate heartworm preventive medication is administered

HELICOBACTER INFECTION



BASICS

OVERVIEW

- *Helicobacter* species are gram-negative, urease-positive bacteria
- The discovery of the association of *Helicobacter pylori* with inflammation of the stomach (known as “gastritis”), stomach ulcers, and stomach cancer has changed the understanding of stomach disease in people
- *Helicobacter* species isolated from stomachs of dogs and cats include *H. felis*, *H. bizzozeronii*, *H. salomonis*, and *Flexispira rappini*
- To date *Helicobacter pylori*, the most important species affecting people, has only been identified in a single colony of laboratory cats
- The cause-effect relationship of *Helicobacter* species with stomach inflammation in dogs and cats is unresolved; inflammation accompanies infection in some, but not all dogs and cats
- The role of *Helicobacter* species in intestinal and liver disease in dogs and cats is unclear
- *Helicobacter canis* has been isolated from both clinically healthy dogs and cats and also in dogs and cats with diarrhea
- *Helicobacter canis* has been isolated from the liver of a puppy with active, multifocal inflammation of the liver (known as “hepatitis”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- None known

Mean Age and Range

- Stomach infection with *Helicobacter* species appears to be acquired at a young age
- The puppy with *Helicobacter canis*-associated inflammation of the liver (hepatitis) was 2 months of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- *Helicobacter* infection without any signs of disease is common
- Vomiting, lack of appetite (known as “anorexia”), abdominal pain, weight loss, and/or rumbling or gurgling sounds caused by movement of gas in the intestinal tract (known as “borborygmus”) have been reported in dogs and cats with *Helicobacter* infections of the stomach
- Diarrhea in dogs may be associated with *Helicobacter canis* infection
- Vomiting, weakness, and sudden death was reported in a dog with *Helicobacter canis* infection of the liver
- May have signs of dehydration from fluid and electrolyte loss due to vomiting and/or diarrhea

CAUSES

Helicobacter Infection of the Stomach

- *H. felis*, *H. heilmannii*, *H. bizzozeronii*, *H. salomonis*, *H. bilis*, and *Flexispira rappini* have been found in dogs
- *H. felis*, *H. pylori*, *H. heilmannii* have been found in cats

Helicobacter Infection of the Intestines and Liver

- *H. fennelliae*—dog (significance unknown)
- *H. cinaedi*—dogs and a cat (significance unknown)
- *H. canis*—in both healthy dogs and cats and in dogs and cats with diarrhea; reported in one dog with sudden (acute) inflammation of the liver (hepatitis)

RISK FACTORS

- Poor sanitary conditions and overcrowding may facilitate spread of infection



TREATMENT

HEALTH CARE

- The ability of *Helicobacter* species to cause disease in dogs and cats is still unclear; therefore, no unanimously accepted guidelines have been adopted for treatment of *Helicobacter* infections in dogs and cats
- Currently animals with *Helicobacter* infection and no clinical signs do not need treatment; this is in sharp contrast to the situation in people, who are treated regardless of symptoms as *Helicobacter pylori* infection is associated with an increased risk for stomach cancer
- Attempt to treat *Helicobacter* infection in dogs and cats with stomach disorders that have compatible clinical signs, which cannot be attributed to another disease process
- Fluid therapy for rehydration

DIET

- Easily digestible diets



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- A triple combination therapy (that is, combination of two antibiotics and one antisecretory drug) is effective in people with *H. pylori* infection with cure rates of approximately 90%
- Combination therapy may eliminate *Helicobacter* infections in dogs and cats less effectively than in people
- Treat for 2 to 3 weeks

DRUG(S) OF CHOICE

Antibiotics (Two Antibiotics with One Antisecretory Agent)

- Possible antibiotics: clarithromycin, metronidazole, amoxicillin, azithromycin, or tetracycline
- Bismuth subsalicylate (original Pepto-Bismol®)

Antisecretory Agents (One with Two Antibiotics)

- Omeprazole, famotidine, ranitidine, or cimetidine

***Helicobacter* Infection in the Intestines and Liver of Dogs**

- Combination of amoxicillin and metronidazole may be effective



FOLLOW-UP CARE

PATIENT MONITORING

- Serologic tests (blood tests that detect the presence of antibodies to a certain disease-causing agent or antigen; an “antibody” is a protein that is produced by the immune system in response to a specific antigen, in this case to *Helicobacter*) are not useful to confirm eradication of the bacteria from the stomach—serum immunoglobulin G (IgG) titers may not decrease for up to 6 months after the infection has been cleared
- ¹³C-urea breath and blood test have been evaluated to monitor the eradication of *Helicobacter* in dogs and cats and show promise for routine application
- If vomiting persists or recurs after cessation of combination therapy, a repeat stomach biopsy to determine whether the infection has been cleared successfully may be necessary

PREVENTIONS AND AVOIDANCE

- Avoid overcrowding and unsanitary conditions

POSSIBLE COMPLICATIONS

- Recurrence
- Zoonotic potential; potential “zoonoses” are diseases that can be passed from animals to people

EXPECTED COURSE AND PROGNOSIS

- The effectiveness of treatment currently employed in dogs and cats for eradicating *Helicobacter* infections is questionable
- Metronidazole, amoxicillin, and famotidine for 14 days effectively eradicated *Helicobacter* in 6 of 8 dogs evaluated 3 days

post-treatment, but all dogs were re-infected by day 28 after completion of treatment

- Clarithromycin, metronidazole, ranitidine, and bismuth for 4 days was effective in eradicating *H. heilmannii* in 11 of 11 cats by 10 days, but two cats were re-infected 42 days post-treatment

- Amoxicillin, metronidazole, and omeprazole for 21 days transiently eradicated *H. pylori* in 6 cats, but all were reinfected 6 weeks post-treatment



KEY POINTS

- Establishing a definitive diagnosis of *Helicobacter* infection is difficult
- *Helicobacter* may be found in normal dogs and cats; the role of *Helicobacter* species in gastrointestinal and liver disease in dogs and cats is unclear
- The effectiveness of treatment currently employed for eradicating *Helicobacter* infections is questionable as reinfection has been seen in many dogs and cats
- *Helicobacter* infections have zoonotic potential; potential “zoonoses” are diseases that can be passed from animals to people

HEMATURIA (BLOOD IN THE URINE)



BASICS

OVERVIEW

- Presence of blood in urine
- Blood in the urine (known as “hematuria”) may indicate a serious underlying disease process

SIGNALMENT/DESCRIPTION of ANIMAL

- Dogs and cats
- Familial hematuria (condition in which blood in the urine runs in certain families of animals) in young animals; cancer in older animals
- Females at greater risk for urinary tract infection (UTI) than are males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Blood in the urine (hematuria) is a sign itself
- Red-tinged urine, with or without abnormal frequent passage of urine (known as “pollakiuria”)
- Mass may be felt during physical examination in patients with cancer
- Abdominal pain, in some patients
- Enlarged and/or painful prostate gland may be felt during physical examination in male dogs
- Bruising (known as “petechiae” if pinpoint spots of bleeding or “ecchymoses” if larger, purplish patches) in patients with blood-clotting disorder (known as “coagulopathy”)

CAUSES

Systemic (Generalized within the Body)

- Blood-clotting disorder (coagulopathy)
- Low number of platelets or thrombocytes in the blood (known as “thrombocytopenia”)
- Inflammation of the blood vessels (known as “vasculitis”)

Upper Urinary Tract (Kidneys and Ureters [Tubes from the Kidneys to the Bladder])

- Structural or anatomic disease, such as cystic kidney disease and familial kidney disease (condition which runs in certain families of animals)
- Metabolic, such as kidney stones (known as “nephrolithiasis”)
- Cancer, such as kidney lymphoma, adenocarcinoma, and hemangiosarcoma
- Infectious disease, such as leptospirosis, feline infectious peritonitis (FIP), and bacterial infection
- Inflammatory disease, such as glomerulonephritis (inflammation of the glomerulus [a tuft of blood capillaries—the “blood filter”] in the kidney)
- Unknown cause (known as “idiopathic disease”)
- Trauma

Lower Urinary Tract (Bladder and Urethra [Tube from the Bladder to the Outside, Through Which Urine Flows Out of the Body])

- Structural or anatomic, such as bladder malformations
- Metabolic, such as stones (known as “uroliths”)
- Cancer, such as transitional cell carcinoma and lymphoma
- Infectious disease, such as bacterial, fungal, and viral disease
- Unknown cause (known as “idiopathic disease”)—cats: feline idiopathic lower urinary tract disease
- Trauma
- Cyclophosphamide-induced hemorrhagic cystitis (a side effect of the chemotherapeutic agent, cyclophosphamide, is a bloody inflammation of the bladder)

Genitalia

- Metabolic, such as the “heat cycle” or “estrus”
- Cancer or tumors, such as transmissible venereal tumor; leiomyoma (tumor that develops from smooth muscle in the urinary tract); and prostate cancer (prostatic adenocarcinoma)

- Infectious disease, such as bacterial and fungal disease
- Inflammatory disease, such as enlargement of the prostate (known as “benign prostatic hyperplasia”) and inflammation of the prostate (known as “prostatitis”)
- Trauma

RISK FACTORS

- Breeds with higher likelihood than other breeds to develop urinary tract stones (known as “urolithiasis”), such as the Dalmatian that forms stones containing urate (known as “urate urolithiasis”); to develop blood-clotting disorders (known as “coagulopathies”), such as the Doberman pinscher that inherits von Willebrand’s disease, a bleeding disorder caused by lower than normal levels of factor VII—one of the ingredients required to clot blood; or to develop cancer, such as the German shepherd dog that has a higher incidence of a cystic kidney cancer (known as “renal cystadenocarcinoma”)



TREATMENT

HEALTH CARE

- Depends on primary or associated diseases
- Urinary tract infection may be associated with another disease involving the urinary tract (known as “local disease”), such as cancer or urinary tract stones (urolithiasis) or involving the body in general (known as “systemic disease”), such as excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”) and diabetes mellitus (“sugar diabetes”) that also requires treatment

DIET

- Urinary tract stones (urolithiasis) and kidney failure may require diet modification

SURGERY

- Surgery may be indicated for cases with urinary tract stones, tumors (cancer), and traumatic injuries to the urinary tract



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Blood transfusion may be necessary if patient has a severely low red-blood cell count (known as “anemia”)
- Fluids to treat dehydration
- Antibiotics to treat urinary tract infection and generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”)
- Heparin for the blood-clotting disorder known as “disseminated intravascular coagulation” or “DIC”



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on primary or associated diseases

PREVENTIONS AND AVOIDANCE

- Depends on primary or associated diseases

POSSIBLE COMPLICATIONS

- Low red-blood cell count (anemia)
- Inadequate or low circulating blood volume (known as “hypovolemia”), if severe bleeding

- Blockage of the one or both ureters (tubes from the kidney to the bladder; known as “ureteral obstruction”) or blockage of the urethra (tube from the bladder to the outside, through which urine flows out of the body; known as “urethral obstruction”) due to blood clots

EXPECTED COURSE AND PROGNOSIS

- Depends on primary or associated diseases



KEY POINTS

- Blood in the urine (known as “hematuria”) may indicate a serious underlying disease process

HEPATIC ENCEPHALOPATHY

(BRAIN DISORDER SECONDARY TO LIVER DISEASE)



BASICS

OVERVIEW

- “Hepatic” refers to the liver; “encephalopathy” is the medical term for any disorder of the brain
- Metabolic disorder affecting the central nervous system, developing secondary to liver disease (known as “hepatopathy”)
- Caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia
- The liver is the largest gland in the body; it has many functions, including production of bile (a fluid substance involved in digestion of fats); production of albumin (a protein in the plasma of the blood); and detoxification of drugs and other chemicals (such as ammonia) in the body
- “Portosystemic shunt” or “portosystemic vascular anomaly” is a condition in which blood vessels allow blood to flow abnormally between the portal vein (vein that normally carries blood from the digestive organs to the liver) and the body circulation without first going through the liver; it can be congenital (present at birth) or acquired (condition that develops sometime later in life/after birth)

GENETICS

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)—inherited in some breeds (such as the Yorkshire terrier, Maltese, cairn terrier, Irish wolfhound, and miniature schnauzer)
- Certain long-term (chronic) diseases of the liver (hepatopathies) have increased likelihood in some breeds (such as the Bedlington terrier, Doberman pinscher, cocker spaniel, Labrador retriever)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)—usually in purebred dogs; increased occurrence in some breeds (such as the Yorkshire terrier, Maltese, cairn terrier, Irish wolfhound, and miniature schnauzer)
- Long-term (chronic) inflammation of the liver (known as “chronic hepatitis”) and condition characterized by abnormal accumulation of copper in the liver, causing liver disease (known as “copper-storage liver disease” or “copper-storage hepatopathy”) are more common in certain breeds (such as the Bedlington terrier, Doberman pinscher, cocker spaniel, Labrador retriever)

Mean Age and Range

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)—usually young animals
- Acquired (condition that develops sometime later in life/after birth) liver disease resulting in acquired portosystemic shunt—any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Nervous system signs—usually associated with meal ingestion, particularly a high-protein meal
- Dramatic temporary resolution of signs may occur with antibiotic or lactulose therapy
- Prolonged recovery from sedation or anesthesia
- Episodic abnormalities or signs may be seen
- Learning disabilities (difficult to train)
- Sluggishness (lethargy) and/or drowsiness or sleepiness (known as “somnolence”)
- Lack of appetite (known as “anorexia”)
- Vomiting
- Disorientation—aimless wandering; compulsive pacing; head pressing
- Increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)
- Blindness related to brain abnormality
- Seizures

- Coma

- Inability to urinate in males that have developed ammonium biurate urinary tract stones (known as “ammonium biurate uroliths”) with subsequent blockage of the urethra (the tube from the bladder to the outside, through which urine flows out of the body; condition known as “obstructive urolithiasis”)

- Excessive salivation (known as “ptyalism”), seizures, aggression, disorientation—more frequent in cats than in dogs

- Compulsive behavior (such as head pressing, circling, aimless wandering, vocalizing); vomiting; diarrhea; increased urination (polyuria) and increased thirst (polydipsia); blood in the urine (known as “hematuria”), frequent voiding of small volumes (known as “pollakiuria”), and difficulty urinating (known as “dysuria”) associated with ammonium biurate urinary tract stones (uroliths)—more frequent in dogs than in cats

- Cats with congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver) may appear normal size, but most have stunted growth or stature; small liver (known as “microhepatica”); and a golden or copper iris (non-blue-eyed and non-Persian cats)

- Dogs with congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly may appear normal size, but usually have stunted growth or stature; small liver (microhepatica)

- Acquired (condition that develops sometime later in life/after birth) liver disease—depends on long-term nature (chronicity) of underlying disorder and formation of acquired portosystemic shunt (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver); fluid build-up in the abdomen (known as “ascites”) is common in dogs with hepatic encephalopathy due to acquired liver disease and often may increase and decrease in severity over time (known as a “waxing and waning” course); variable bleeding

- Lower urinary tract signs (such as straining to urinate; difficulty urinating); orange/brown color to urine due to presence of ammonium biurate crystals in the urine; inability to urinate in males that have developed ammonium biurate urinary tract stones (ammonium biurate uroliths) with subsequent blockage of the urethra (the tube from the bladder to the outside, through which urine flows out of the body; condition known as obstructive urolithiasis)

CAUSES

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)—malformations of blood vessels during development of circulation

- Acquired (condition that develops sometime later in life/after birth) portosystemic shunt—occurs with diseases that lead to high blood pressure in the portal vein (the vein carrying blood from the digestive organs to the liver; high blood pressure in the portal vein is known as “portal hypertension”), such as progressive damage and scarring of the liver (known as “cirrhosis”)

- Sudden (acute) liver failure—induced by drugs, toxins, or infection

RISK FACTORS

- Alkalosis (condition in which the pH of the body is too high)

- Decreased levels of potassium in the blood (known as “hypokalemia”)

- Certain anesthetics and sedatives

- Certain medications (such as methionine, tetracycline, antihistamines)

- Bleeding into the intestine—most common cause that leads to signs of hepatic encephalopathy (blood acts as a source of protein, which when broken down forms ammonia)

- Transfusion—stored blood products containing high concentrations of ammonia; incompatible blood transfusions

- Infections

- Constipation

- Breakdown of lean muscle mass and body tissues (known as “catabolism”)—disorders causing muscle wasting; normally large amounts of ammonia are detoxified temporarily by storage in muscle



TREATMENT

HEALTH CARE

- Depends on underlying condition; eliminate factors promoting hepatic encephalopathy

- Improve dietary protein tolerance

- Discontinue medications administered by mouth, if pet is comatose (known as a “hepatic coma”)

- Avoid risk factors

- Fluids—if pet has low blood glucose or sugar (known as “hypoglycemia”), supplement fluids with 2.5% to 5% dextrose; provide

potassium chloride, according to needs; restrict sodium in fluids for pets with acquired (condition that develops sometime later in life/after birth) liver disease, fluid build-up in the abdomen (ascites), and/or marked low levels of albumin (a protein) in the body (condition known as “hypoalbuminemia”)

- B vitamins

ACTIVITY

- Keep patient warm, inactive, and hydrated

DIET

- Adequate calories—avoid breakdown of lean muscle mass and body tissues (catabolism) and maintain muscle mass (as a site for temporary ammonia detoxification/storage)
- Dietary protein restriction—cornerstone of medical management; use commercially formulated diet specific for liver disease or kidney disease, as directed by your pet’s veterinarian; dogs: dairy and soy protein best sources; cats: as pure carnivores, they must have meat-derived protein
- Good-quality vitamin supplements (without methionine)—vitamin usage in the body is changed with liver disease and vitamins are lost in urine
- Ensure adequate thiamine (a B vitamin)
- Nutrition through intravenous route (known as “parenteral nutrition”)—may be used as a partial means of providing nutrition (recommended for short-term lack of appetite) to minimize breakdown of muscle (catabolism) or as a total means of providing nutrition (known as “total parenteral nutrition” or “TPN”), which is recommended if the pet has lack of appetite of more than 5 days and providing nutrition via the gastrointestinal tract is not possible

SURGERY

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)—surgical correction in which abnormal blood vessels are “tied off” or “ligated” is a possibility in many cases (potential exists that only some of the blood vessels can be ligated)
- Acquired (condition that develops sometime later in life/after birth) portosystemic shunt—abnormal blood vessels should not be “tied off” or “ligated”



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medications that increase dietary protein tolerance, change bacteria or conditions in the intestines, reduce production or availability of substances provoking hepatic encephalopathy
- Antibiotics—antibiotic selection based on ability to change the bacteria in the intestines or their products; first choice antimicrobial selections: administered by injection (known as “systemic administration”), such as metronidazole or amoxicillin; combine use with lactulose
- Nonabsorbable-fermented carbohydrates—lactulose, lactitol, or lactose (if lactase deficient); decrease production or absorption of ammonia; increase rate of stool transit; trap nitrogen in bacteria; lactulose most commonly used; therapeutic goal is passage of two to three soft stools daily; also may be administered as an enema for sudden (acute) hepatic encephalopathy and coma *after* cleansing enemas have removed debris
- Enemas—*cleansing enemas* (warmed polyionic fluids) mechanically clean colon; *retention enemas* directly deliver fermentable substrates or directly alter colonic pH and organisms: diluted lactulose, lactitol, or lactose; neomycin in water; diluted Betadine®
- Zinc supplementation, as directed by your pet’s veterinarian
- Fluid build-up in the brain (known as “cerebral edema”)—complicates sudden (acute) hepatic encephalopathy; administer medication (mannitol) to decrease fluid build-up; administer nasal oxygen and *N*-acetylcysteine; use of steroids to decrease fluid build-up (edema) is controversial as steroids may promote bleeding in the intestinal tract (which is a risk factor)
- If epileptic seizure activity—zonisamide or potassium bromide is the preferred medication to control seizures (known as “anticonvulsants”) compared to phenobarbital



FOLLOW-UP CARE

PATIENT MONITORING

- Re-evaluate patient's at-home behavior, demeanor, body condition, and weight
- Monitor albumin and glucose—in patients with non-correctable disorders; adjust nutrition
- Monitor electrolytes—especially potassium; avoid low levels of potassium in the blood (hypokalemia) as it aggravates increased levels of ammonia in the blood (known as “hyperammonemia”)

PREVENTIONS AND AVOIDANCE

- Avoid dehydration; excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”); breakdown of red-blood cells (known as “hemolysis”); constipation; bleeding into the intestines; internal parasites; transfusion of stored blood; ammonium challenge; urinary tract infections; low levels of potassium in the blood (hypokalemia); low levels of magnesium in the blood (known as “hypomagnesemia”); and high pH in the blood (known as “alkalemia”)

POSSIBLE COMPLICATIONS

- Permanent nervous system damage (rare)
- Complications from surgical ligation of abnormal blood vessels in cases with congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)
- Liver failure
- Death

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying disorder
- Sudden (acute) or long-term (chronic) liver failure—may be fully or partially reversible, or patient may die



KEY POINTS

- Hepatic encephalopathy—often episodic and relapsing, if underlying disorder cannot be cured
- Learn to administer enemas and to adjust the dose of medications carefully, as directed by your pet's veterinarian
- Congenital (present at birth) portosystemic shunt or portosystemic vascular (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)—surgical ligation may be curative, but ligation can cause complications in some dogs; postoperative clinical signs may persist requiring long-term (chronic) nutritional and medical management

HEPATIC LIPIDOSIS

(ACCUMULATION OF FATS AND LIPIDS IN THE LIVER)



BASICS

OVERVIEW

- Disease in which fats and lipids (compounds that contain fats or oils) accumulate in the liver (condition known as “hepatic lipidosis”)
- Possible complication of lack of appetite (known as “anorexia”) in obese cats
- Feline hepatic lipidosis—more than 50% of liver cells (known as “hepatocytes”) accumulate triglycerides, results in severe decrease or stoppage of the flow of bile (known as “cholestasis”) and liver dysfunction in the cat
- Usually secondary to another underlying disease process or simply lack of food intake (such as a cat accidentally being locked in basement)
- The liver is the largest gland in the body; it has many functions, including production of bile (a fluid substance involved in digestion of fats); production of albumin (a protein in the plasma of the blood); and detoxification of drugs and other chemicals (such as ammonia) in the body
- Bile ducts begin within the liver itself as tiny channels to transport bile—the ducts join together to form larger bile ducts and finally enter the extrahepatic or common bile duct, which empties into the upper small intestine; the system of bile ducts is known as the “biliary tree”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats primarily affected
- Dogs rarely affected (puppies, especially with storage disease as in the Maltese; “storage disease” is an inherited metabolic disease in which harmful levels of materials accumulate in the body’s cells and tissues)

Breed Predilection

- None

Mean Age and Range

- Mean—8 years of age (range, 1 to 16 years of age)
- Primarily middle-aged adults

Predominant Sex

- Inconsistent susceptibility to develop hepatic lipidosis for obese females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Lack of appetite (anorexia) and weight loss
- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)
- Sluggishness (lethargy)
- Weakness progressing to collapse
- Vomiting, diarrhea, or constipation
- Excessive salivation (known as “ptyalism”), may reflect hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia) or food aversion
- Abnormal position of the head and neck, in which the chin is located near the chest (known as “neck ventroflexion”)
- Enlargement of the liver (known as “hepatomegaly”)
- Dehydration
- Abnormalities due to underlying diseases

CAUSES

- More than 85% cats with hepatic lipidosis have disorders causing lack of appetite (anorexia) or problems absorbing nutrients into the body following digestion (known as “malassimilation”)
- Primary liver disease—portosystemic shunt (condition in which blood vessels allow blood to flow abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver); inflammation of the bile duct or biliary tree (known as “cholangitis”) and inflammation of the bile ducts and liver (known as “cholangiohepatitis”); blockage of the extrahepatic or common bile duct (known as “extrahepatic bile duct obstruction”); presence of hard, solid material in the bile duct or gall bladder (known as “cholelithiasis”); cancer

- Gastrointestinal disease—blockage or obstruction of the gastrointestinal tract; cancer (such as lymphoma, a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body); inflammatory bowel disease (IBD); inflammation of the pancreas (known as “pancreatitis”)
- Urinary tract disease—long-term (chronic) inflammation of the tissue spaces in the kidneys (known as “chronic interstitial nephritis”); lower urinary tract infection; kidney failure
- Nervous system disorders
- Infectious diseases—toxoplasmosis; feline infectious peritonitis (FIP); feline immunodeficiency virus (FIV) or feline leukemia virus (FeLV)-related disease
- Excess levels of thyroid hormone (known as “hyperthyroidism”)
- B₁₂ deficiency (may increase susceptibility to develop hepatic lipidosis in cats)
- Many other generalized (systemic) conditions or toxins

RISK FACTORS

- Obesity
- Lack of appetite (anorexia)
- Breakdown of lean muscle mass and body tissues (known as “catabolism”)
- Rapid weight loss
- B₁₂ deficiency



TREATMENT

HEALTH CARE

- Inpatient—necessary for cats that have yellowish discoloration to the gums and other tissues of the body (jaundice or icterus); abnormal position of the head and neck, in which the chin is located near the chest (neck ventroflexion; caused by muscular weakness which can be due to severe electrolyte disturbance of potassium or phosphate, or to thiamine [a B vitamin] deficiency)
- Home care after stabilization and feeding tube has been placed and is functioning problem free
- Outpatient—reduces stress and thereby facilitates recovery in some cats
- Balanced fluids
- Potassium supplementation is important
- Phosphate supplements usually needed
- Magnesium supplements sometimes needed

ACTIVITY

- Cats recently diagnosed or early in recovery phase of hepatic lipidosis may be too weak for any activity
- Activity may help to improve motility of the stomach, when partial paralysis of the muscles of the stomach (known as “gastroparesis”) complicates feeding

DIET

- Nutritional support—cornerstone of recovery
- High-protein, high-calorie diet is essential
- Energy—60 to 90 kcal/kg ideal body weight/day
- Forced feeding of some type usually is required; however, forced feeding by mouth may lead to food aversion
- Tube feeding; initially by tube placed into the nose, down the esophagus (the tube from the mouth to the stomach) and into the stomach (known as a “nasogastric tube”) and then an esophageal tube after corrected hydration and electrolyte status, and administration of vitamin K₁
- Avoid surgery to place a stomach feeding tube, as cats with hepatic lipidosis have high mortality
- Cautiously offer food daily to assess interest in food and appetite
- Human “stress-formula” intestinal diets generally are not recommended—require supplemental amino acids (arginine [or citrulline], and taurine)—amino acids are the smallest components of protein; taurine is an amino acid that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the retina (back of the eye) and heart
- Supplements—improve survival in severely affected cats: L-carnitine; taurine; thiamine (a B vitamin); vitamin B₁₂; water-soluble vitamins (vitamin B, vitamin C); vitamin E; thiol donors (such as S-adenosyl-L-methionine [SAME]); potassium gluconate (for low

levels of potassium in the blood [known as “hypokalemia”]), reduces fluid potassium supplements; marine oil in food

- Carnitine supplements have wide variability in bioavailability; Carnitor™ (liquid medical grade carnitine) is recommended

SURGERY

- Exploratory surgery and liver biopsy (if indicated)—inspect for underlying disorders; possibly biopsy the pancreas, stomach, and/or small bowel
- Avoid surgical interventions until hydration, electrolyte depletions, and any blood abnormalities are corrected



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Vitamin K₁—recommended for all cats suspected of having hepatic lipidosis
- Drugs to decrease signs of hepatic encephalopathy (such as drooling, seizures, aggression, and disorientation) usually are not needed; hepatic encephalopathy is a brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia
- Metoclopramide—for vomiting, nausea, and partial paralysis of the muscles of the stomach (gastroparesis)
- Systemic antibiotics—as appropriate for coexistent infections
- S-adenosyl-L-methionine (S-AMe; Denosyl-SD4®)



FOLLOW-UP CARE

PATIENT MONITORING

- Frequent reevaluations—imperative
- Body weight, condition, hydration, electrolytes
- Judicious adjustments of energy, fluids, and electrolyte provisions—important
- Serum bilirubin—“bilirubin” is a normal bile pigment formed from the breakdown of hemoglobin; “hemoglobin” is the compound in red-blood cells that carries oxygen to the tissues of the body; the liver takes up the hemoglobin following normal or abnormal breakdown of red-blood cells and processes it to form bile (a fluid substance involved in digestion of fats), which provides a means of eliminating bilirubin from the body; bilirubin levels in the blood can increase if the flow of bile is blocked—in the case of hepatic lipidosis, bilirubin is not eliminated from the body at a normal rate, so they increase—serum bilirubin levels decline within 2 weeks of adequate medical management of hepatic lipidosis and predict recovery
- Liver enzyme activity levels are slow to return to normal; do not predict recovery
- Discharge for home care—when vomiting is controlled, partial paralysis of the muscles of the stomach (gastroparesis) has resolved, serum bilirubin levels are declining, patient is able to walk around (known as being “ambulatory”), and tube-feeding apparatus has been problem-free
- Tube feeding—discontinued only after confirmed voluntary food consumption for 2 weeks

PREVENTIONS AND AVOIDANCE

- Obesity—prevent; weight reduction must not exceed 2% body weight per week
- Caution owner to verify food intake during weight loss regimens and during at-home stress

POSSIBLE COMPLICATIONS

- Feeding tube malfunction or obstruction—tube blockage or obstructions may be relieved by papaya juice, carbonated soft drink, or pancreatic enzyme slurry, as directed by your pet’s veterinarian
- Rare to have hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia) develop after dietary support introduced
- Liver failure, leading to death
- Untreatable underlying cause

EXPECTED COURSE AND PROGNOSIS

- Untreated—progressive disease and death

- Optimal response to tube feeding and nutritional supplements—recovery in 3 to 6 weeks
- Treatment as described— 85% recovery of severely affected animals
- Underlying disease influences outcome
- Hepatic lipidosis rarely recurs
- Hepatic lipidosis does not cause long-term (chronic) liver dysfunction



KEY POINTS

- Sequential blood work (serum biochemical assays) needed to assess recovery
- Learn use and care of feeding tube
- Feeding tubes may be retained for 4 to 6 months
- Underlying disease influences outcome
- Hepatic lipidosis rarely recurs
- Hepatic lipidosis does not cause long-term (chronic) liver dysfunction

CHRONIC ACTIVE HEPATITIS

(LONG-TERM, ON-GOING INFLAMMATION OF THE LIVER)



BASICS

OVERVIEW

● Long-term (known as “chronic”), ongoing (known as “active”) inflammation of the liver (known as “hepatitis”) associated with accumulation of inflammatory cells and progressive scarring or formation of excessive fibrous tissue (known as “fibrosis”)

GENETICS

- Inherited copper-storage disease of the liver—Bedlington terriers; other breeds
- May play a role in chronic, active hepatitis seen in cocker spaniels, Doberman pinschers, Labrador retrievers

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog

Breed Predispositions

- Bedlington terriers
- Doberman pinschers
- Cocker spaniels
- Labrador retrievers
- Skye terriers
- Standard poodles
- West Highland white terriers (?)

Mean Age and Range

- Mean age—6 years
- Range—2 to 10 years of age

Predominant Sex

- Many breeds—females may be at higher risk than males
- Cocker spaniels—more common in males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sluggishness (lethargy)
- Lack of appetite (known as “anorexia”)
- Weight loss
- Vomiting
- Excessive urination (known as “polyuria”) and excessive thirst (known as “polydipsia”)
- Yellowish discoloration to gums, moist tissues of body (known as “mucous membranes”) and other tissues (discoloration known as “icterus” or “jaundice”)
- Fluid build-up in the abdomen (known as “ascites”)
- Poor body condition
- Nervous system signs (such as dullness, seizures) caused by accumulation of ammonia in the system, due to inability of the liver to rid the body of ammonia (condition known as “hepatic encephalopathy”)

CAUSES

- Infectious disease—canine hepatitis virus; leptospirosis; canine acidophil-cell hepatitis (viral infection hypothesized, but may be initiated by other infectious agents)
- Immune-mediated disease—autoimmune disease
- Toxic—copper-storage disease; drugs (such as anticonvulsants, trimethoprim-sulfa [antibiotic], dimethylnitrosamine, oxbendazole); environmental

RISK FACTORS

- Breed
- Age
- Gender
- Drugs, especially anticonvulsants



TREATMENT

HEALTH CARE

- Inpatient—for diagnostic testing and initiation of medical therapy in overtly ill dogs
- Outpatient—if condition is stable at diagnosis
- Depends on underlying condition
- Fluid therapy—balanced electrolyte fluids, supplemented appropriately with potassium and dextrose; electrolytes are chemical compounds (such as sodium, potassium, chloride) necessary for the body to function; restrict sodium if fluid build-up in the abdomen (ascites) is present
- B-vitamins
- Drugs to increase elimination of fluids from the body (known as “diuretics”) are the first option to decrease fluid build-up in the abdomen (ascites)
- Tapping the abdomen to withdraw or drain excessive fluid (known as “abdominocentesis”)—sterile procedure used when fluid build-up in the abdomen is causing problems with food intake and/or breathing or impairing sleep

ACTIVITY

- Keep patient warm, inactive, and hydrated
- Rest and inactivity may promote healing of the liver; normal blood glucose (sugar) levels (known as “euglycemia”), and elimination of fluid build-up in the abdomen (ascites)

DIET

- Adequate calories—maintain muscle mass and body weight; record body condition score
- Dietary protein—restrict only if animal has nervous system signs (such as dullness, seizures) caused by accumulation of ammonia in the system, due to inability of the liver to rid the body of ammonia (hepatic encephalopathy); feed balanced diet; with hepatic encephalopathy, avoid fish, meat, and egg-quality protein (dogs); cats are true carnivores and require meat-source protein
- Meal frequency—feeding several small meals per day improves use of nutrients by the body
- Sodium restriction—with fluid build-up in the abdomen (ascites) or severely low levels of albumin (a protein) in the blood (known as “hypoalbuminemia”)
- Good-quality vitamin supplement (without methionine)—vitamin metabolism is disturbed with liver disease and vitamins are lost into the urine—the animal needs vitamin supplementation to counter the disturbed metabolism and loss of vitamins into the urine
- Thiamine
- Partial intravenous feeding (known as “parenteral nutrition”)—recommended for short-term lack of appetite: give total parenteral nutrition if lack of appetite lasts more than 5 days

SURGERY

- Surgical repair of acquired portosystemic shunt (condition of abnormal blood flow in the liver due to high blood pressure in the portal vein, the vein carrying blood from the digestive organs to the liver)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Diuretics (drugs to increase elimination of fluids from the body)

- For fluid build-up in the abdomen (ascites)—combination of furosemide and spironolactone; recheck and adjust dose at 4 to 7-day intervals

Drugs for Hepatic Encephalopathy (nervous system disorder with signs [such as dullness, seizures] caused by

accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)

- Antibiotics
- Nonabsorbable-fermented carbohydrates (such as lactulose) to decrease production of ammonia and to decrease absorption of ammonia from the intestinal tract into the body
- Enemas to clean out the large bowel or colon
- Zinc supplementation
- Drugs to decrease swelling in the brain, if present; example, mannitol

Antioxidants

- Vitamin E— α -tocopherol
- S-adenosylmethionine (S-AdoMet)
- Avoid vitamin C (ascorbate) with high tissue-copper or iron concentration—augments oxidant injury associated with transition metals

Zinc (Zinc Acetate)

- Antioxidant and antifibrotic effects (“antifibrotic” refers to stopping or preventing formation of excessive fibrous tissue [fibrosis])
- Blocks copper uptake from the intestinal tract

Copper Chelation (use of specific chemicals to tie up copper in the system and to allow it to be removed from the body)

- D-Penicillamine (first choice) or trientine
- D-Penicillamine chelates copper and promotes excretion of copper into the urine and is suspected to have other liver protective effects; treatment should be initiated in affected dogs having abnormal liver-copper concentrations
- Follow chelation therapy with long-term zinc supplementation

Immunomodulation (drugs that alter the immune response)

- Steroids, such as prednisolone or prednisone; if animal has fluid build-up in the abdomen (ascites), use dexamethasone to avoid steroid influence on sodium retention in the body (known as “mineralocorticoid effect”) seen with prednisolone and prednisone
- Azathioprine—additional therapy for immune-mediated inflammation
- Mycophenolate mofetil (very limited experience)—for patients that do not tolerate azathioprine
- Microemulsified cyclosporine—option, but limited long-term experience
- Ursodeoxycholic acid—modifies the immune response; provides protective effect to liver; prevents formation of excessive fibrous tissue; antioxidant

Antifibrotics (drugs that prevent formation of excessive fibrous tissue [fibrosis])

- Polyunsaturated phosphatidylcholine (phosphatidylcholine lecithin)—steroid-sparing effect allows lower dosage of prednisolone for disease management; other effects: modifies the immune response; acts as an antioxidant; provides protective effect to the liver
- Colchicine—inhibits collagen production
- Silibinin—protects the liver from numerous toxins; has antifibrotic and antioxidant effects; promotes liver-cell regeneration; especially if toxin- or drug-mediated injury suspected



FOLLOW-UP CARE

PATIENT MONITORING

- At-home behavior
- Body condition and weight—adjust food intake to maintain weight
- Complete blood count (CBC), serum biochemistry profile, and urinalysis—look for signs of drug toxicity and disease remission
- Patients receiving azathioprine should be monitored by blood work (including complete blood count [CBC] and biochemistry profile) every 7 to 10 days for first month to ensure absence of bone-marrow, liver, and pancreatic toxicity; if sudden (acute) bone-marrow toxicity occurs, stop therapy, allow recovery, then reintroduce drug at lower dose; if long-term (chronic) bone-marrow toxicity (after many months) or sudden (acute) cholestatic liver disease (disease in which the flow of bile is decreased or stopped) or pancreatic injury are identified, discontinue therapy permanently
- Patients receiving mycophenolate mofetil should be monitored for bone-marrow toxicity (rare at recommended dose)
- Patients receiving colchicine should be monitored for bloody diarrhea and bone-marrow suppression (adverse side effects of the drug)

PREVENTIONS AND AVOIDANCE

- Maintain high vigilance for early signs of inflammation of the liver (hepatitis) in breeds that are more likely to develop liver disease than other breeds; early signs of liver inflammation include high liver-enzyme activity, as seen on blood work

- Determine diagnosis and initiate therapy early

POSSIBLE COMPLICATIONS

- Sepsis (presence of pus-forming bacteria and their poisons in the blood or tissues) secondary to the animal's inability to develop a normal immune response
- Nervous system signs (such as dullness, seizures) caused by accumulation of ammonia in the system, due to inability of the liver to rid the body of ammonia (hepatic encephalopathy)
- Blood-clotting disorder (disseminated intravascular coagulopathy or DIC)
- Intestinal ulceration
- Liver failure and death

EXPECTED COURSE AND PROGNOSIS

- Depends on underlying disorder
- Long-term (chronic) disease; yellowish discoloration to gums, moist tissues of body (known as "mucous membranes") and other tissues (icterus or jaundice); and fluid build-up in the abdomen (ascites)—poorer prognosis
- Use of multiple drugs (known as "polypharmacy") and nutritional support extend quality survival, compared with untreated cases, but no long-term studies yet available
- Early diagnosis in Doberman pinschers, Bedlington terriers, and cocker spaniels appears to delay disease progression for years



KEY POINTS

- Medication is required for life; disease is cyclic
- Quarterly or biannual physical examinations and evaluations are needed
- Lack of long-term veterinary studies to prove effectiveness of single or multiple (polypharmacy) drug approaches; recommendations derived from (1) broad clinical experience, (2) retrospective and prospective studies in human beings, and (3) animal disease models

VACUOLAR HEPATOPATHY

(DISORDER CHARACTERIZED BY THE PRESENCE OF CAVITIES [VACUOLES] WITHIN LIVER CELLS)



BASICS

OVERVIEW

- “Vacuolar” refers to vacuoles; “vacuoles” are small cavities within cells—the cavities are surrounded by a membrane and they contain various substances (such as fluids, storage products, or waste products)
- “Hepatopathy” is the medical term for a disorder or disease of the liver
- Vacuolar hepatopathy—reversible change in the liver cells of dogs; associated with accumulation of glycogen in clear cavities within liver cells; “glycogen” is the material that serves as the carbohydrate storage or reserve of the body, which is broken down readily into glucose (sugar)
- Development of the clear cavities (vacuoles) containing glycogen often is secondary to steroid treatment; increased levels of steroids in the body (either produced by the adrenal glands [known as “spontaneous hyperadrenocorticism” or “spontaneous Cushing’s disease”] or through administration of steroid-containing medications [known as “iatrogenic hyperadrenocorticism” or “iatrogenic Cushing’s disease”]); enlargement of the adrenal glands (known as “adrenal hyperplasia”) leading to release of adrenal sex hormones [especially progesterone]); or long-term (chronic) illness of other systems
- Condition leads to high alkaline phosphatase (ALP) activity as seen on blood tests, often without signs of liver insufficiency; “alkaline phosphatase” is a normal enzyme found in several types of cells, including liver cells; increased levels of alkaline phosphatase may indicate abnormal liver function
- In some animals, vacuolar hepatopathy is associated with the presence of lipids (compounds that contain fats or oils) in the clear cavities—may occur in animals with high levels of lipids in their blood for unknown reason (so called “idiopathic hyperlipidemia”); lipid with glycogen—diabetes mellitus (“sugar diabetes”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs; rarely cats

Breed Predispositions

- Breeds susceptible to increased levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing’s disease), such as the miniature poodle, dachshund, boxer, Boston terrier
- Enlargement of the adrenal glands with increased levels of adrenal sex hormones (sex-hormone adrenal hyperplasia) and increased levels of lipids (compounds that contain fats or oils) in the blood (hyperlipidemia)—Scottish terrier
- Increased levels of lipids (compounds that contain fats or oils) in the blood (hyperlipidemia)—miniature schnauzer

Mean Age and Range

- Middle-aged to old dogs—more than 75% of dogs with increased levels of steroids produced by the adrenal glands (spontaneous hyperadrenocorticism) are older than 9 years of age; long-term (chronic) inflammation or tumors
- Dogs of any age—vacuolar hepatopathy subsequent to steroid administration (known as “iatrogenic vacuolar hepatopathy”)
- Young dogs or cats—genetic increased levels of lipids (compounds that contain fats or oils) in the blood (hyperlipidemia)
- Puppies or kittens—disease in which fats and lipids (compounds that contain fats or oils) accumulate in the liver (known as “juvenile hepatic lipidosis”)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs often related to effects of steroids or other generalized (systemic) illnesses causing stress
- Rarely, signs of liver disease or failure; however, liver failure can occur with severe, long-term (chronic) vacuolar hepatopathy
- Signs related to high levels of steroids—increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”); increased appetite (known as “polyphagia”); hair loss (known as “alopecia”); abdominal distention; muscle weakness; panting; sluggishness (lethargy); fragile skin; bruising
- Enlargement of the adrenal glands with increased levels of adrenal sex hormones (sex-hormone adrenal hyperplasia)—similar signs to those of high levels of steroids, but may be fewer and less severe; hair loss (alopecia) with darkened skin (known as “hyperpigmentation”) is least common; some dogs have no signs and only finding is chronically high alkaline phosphatase; “alkaline phosphatase” is a normal enzyme found in several types of cells, including liver cells; increased levels of alkaline phosphatase may indicate abnormal liver function
- Enlarged liver (known as “hepatomegaly”)
- Other signs relate to the underlying disease

CAUSES

- Steroid administration
- Increased levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing's disease)
- Enlargement of the adrenal glands with increased levels of adrenal sex hormones (sex-hormone adrenal hyperplasia)—over-production of various steroid hormones (especially progesterone)
- Generalized (systemic) diseases associated with stress—examples include severe dental disease; inflammatory bowel disease (IBD); long-term (chronic) inflammation of the pancreas (known as “pancreatitis”); generalized cancer (especially lymphoma; “lymphoma” is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body); long-term (chronic) infections (urinary tract, skin); low levels of thyroid hormone (known as “hypothyroidism”); errors of lipid (compound that contains fats or oils) metabolism, leading to lipid or glycogen accumulation

RISK FACTORS

- Treatment with steroid-containing medications
- Breeds at risk for increased levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing's disease), such as poodles, dachshunds, Boston terriers, and beagles
- Breeds at risk for increased levels of lipids (compounds that contain fats or oils) in the blood (hyperlipidemia), such as miniature schnauzers, Shetland sheepdogs, beagles



TREATMENT

HEALTH CARE

- Outpatient—common for underlying disease

ACTIVITY

- Normal

DIET

- Fat restriction for cases with increased levels of lipids (compounds that contain fats or oils) in the blood (hyperlipidemia) or inflammation of the pancreas (pancreatitis)
- Cautious calorie restriction for obese animals, as directed by your pet's veterinarian

SURGERY

- Depends on underlying conditions
- Adrenal gland masses may be removed surgically in some patients
- Pituitary gland masses may be removed surgically, but only by surgeons experienced in the procedure; pituitary gland masses may be better treated with radiation



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Depend on the underlying disease
- Increased production of steroids by the adrenal glands secondary to stimulation from the pituitary gland (known as “pituitary-dependent hyperadrenocorticism”) or enlargement of the adrenal glands with increased levels of adrenal sex hormones (sex-hormone adrenal hyperplasia)—usually treated medically once diagnosis confirmed; treatment may include op'-DDD (Lysodren®), ketoconazole, or trilostane; l-deprenyl usually ineffective; op'DDD (Lysodren®) preferred for sex-hormone adrenal hyperplasia; melatonin appears to be ineffective for treating sex-hormone adrenal hyperplasia
- Management of generalized (systemic) inflammatory disorders that necessitate the use of medications to decrease the immune response (known as “immunosuppressive drugs”) or medications to decrease inflammation (known as “anti-inflammatory drugs”)—use multiple medications to avoid or minimize steroid exposure in patients with signs of vacuolar hepatopathy; medications selected may include metronidazole (for treatment of inflammatory bowel disease); chemotherapeutic drugs used to decrease the immune response (such as azathioprine, chlorambucil, cyclophosphamide); and other immunosuppressive drugs (such as mycophenolate, cyclosporine)

- Cancer—chemotherapy or radiation, as appropriate
- Dental disease—antibiotics and dentistry
- Infection/inflammation of the kidney (known as “pyelonephritis”); long-term (chronic) inflammation of the skin (known as “dermatitis”); or other infectious disorders—long-term (chronic) antibiotic treatment, based on bacterial culture and sensitivity tests; other appropriate medications
- Low levels of thyroid hormone (hypothyroidism)—supplemental thyroid hormone



FOLLOW-UP CARE

PATIENT MONITORING

- Enlarged liver (hepatomegaly)—feeling the abdomen and liver during physical examination (known as “abdominal palpation”); X-rays or ultrasound examination; blood work (serum biochemical profile to evaluate improvement of liver enzyme levels)
- Adrenal gland function—ACTH-stimulation test, to determine response of the adrenal gland
- Cancer—physical examination and diagnostic imaging (X-rays, ultrasound examination, computed tomography [CT or CAT scan], magnetic resonance imaging [MRI])
- Control of infection—repeat bacterial cultures
- Increased levels of lipids (compounds that contain fats or oils) in the blood (hyperlipidemia)—assess presence of lipids visible in the blood (known as “gross lipemia”); measure triglycerides and cholesterol levels

PREVENTIONS AND AVOIDANCE

- Limit steroids for treatment of confirmed conditions that require steroid therapy
- When steroids are necessary, use alternate-day therapy (if possible) with prednisone; titrate steroids to lowest effective dose (as directed by your pet’s veterinarian)

POSSIBLE COMPLICATIONS

- Numerous complications related to the effects of steroids and associated conditions are possible; discuss possible complications with your pet’s veterinarian

EXPECTED COURSE AND PROGNOSIS

- Most patients do not have signs of liver disease, despite high alkaline phosphatase; however, progressive liver disease (hepatopathy) leading to widespread (diffuse) nodule formation and liver failure may occur in long-term (chronic) vacuolar hepatopathy in patients only showing long-term (chronic) markedly high alkaline phosphatase levels; “alkaline phosphatase” is a normal enzyme found in several types of cells, including liver cells
- Blood work and liver abnormalities associated with vacuolar hepatopathy are completely reversible before liver nodules form



KEY POINTS

- Vacuolar hepatopathy—reversible change in the liver cells of dogs; associated with accumulation of glycogen in clear cavities within liver cells; “glycogen” is the material that serves as the carbohydrate storage or reserve of the body, which is broken down readily into glucose (sugar)
- Most patients do not have signs of liver disease, despite high alkaline phosphatase; however, progressive liver disease (hepatopathy) leading to widespread (diffuse) nodule formation and liver failure may occur in long-term (chronic) vacuolar hepatopathy in patients only showing long-term (chronic) markedly high alkaline phosphatase levels; “alkaline phosphatase” is a normal enzyme found in several types of cells, including liver cells
- Blood work and liver abnormalities associated with vacuolar hepatopathy are completely reversible before liver nodules form
- Limit steroids for treatment of confirmed conditions that require steroid therapy
- When steroids are necessary, use alternate-day therapy (if possible) with prednisone; titrate steroids to lowest effective dose (as directed by your pet’s veterinarian)

LIVER TOXINS (HEPATOTOXINS)



BASICS

OVERVIEW

- Liver toxins are substances (such as drugs or toxins) that cause liver injury
- Direct liver toxin—cause predictable injury to liver cells and the liver
- Individual pet may be more likely to develop ill effects to a particular medication than other animals and the reaction is unexpected (known as “idiosyncratic reactions”)—unpredictable injury
- The liver is the largest gland in the body; it has many functions, including production of bile (a fluid substance involved in digestion of fats); production of albumin (a protein in the plasma of the blood); and detoxification of drugs and other chemicals (such as ammonia) in the body

GENETICS

- Some breeds of dogs may be susceptible to certain drug-associated liver toxicities

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Dogs may be more susceptible to certain liver toxins than other mammals
- Cats have higher risk than dogs to some liver toxins

Breed Predispositions

- Some dog breeds have high risk for selected drug toxicities—examples include Doberman pinschers, Dalmatians, Samoyeds: trimethoprim-sulfa (an antibiotic); Doberman pinschers: oxbendazole (medication used to kill intestinal parasites); Labrador retrievers: possibly nonsteroidal anti-inflammatory drugs (NSAIDs); German shepherd dogs: phenobarbital
- Siamese cats—some families or lines have high risk

Mean Age and Range

- Any age
- Young animals (less than 16 weeks of age)—immature liver and less ability to detoxify drugs and other chemicals; more likely to eat substances that are potentially toxic

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs may reflect long-term (chronic) exposure or single, sudden (acute) exposure to a liver toxin
- Severe general signs of discomfort and “not feeling well” (known as “malaise”) to near death (known as the “moribund state”)
- Gastrointestinal signs: lack of appetite (known as “anorexia”), vomiting, diarrhea
- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)
- Variable fever
- Weakness
- Fluid build-up in the abdomen (known as “ascites”)—rare (grave sign)
- Severe liver failure—brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”) or coma
- Blood-clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”) secondary to the death of liver tissue (known as “liver necrosis”)—bleeding; small, pinpoint areas of bleeding (known as “petechia”); bruises or purplish patches under the skin, due to bleeding (known as “ecchymoses”)

CAUSES

Many substances (such as drugs or toxins) may cause liver toxicity, including some drugs that are used routinely in the treatment of dogs and cats. Examples include the following:

Commonly Reported Drugs

- Azole antifungals
- Amoxicillin (an antibiotic)
- Azathioprine (a chemotherapeutic drug, frequently used to decrease the immune response)
- Nonsteroidal anti-inflammatory drugs (NSAIDs)—dogs

- CCNU—dogs
- Cyclosporine (used to decrease the immune response)
- Diazepam (used to decrease anxiety, to control seizures, and as an appetite stimulant)—cats
- Acetaminophen (pain reliever and fever reducer)—dogs and cats (note: acetaminophen should never be administered to cats as it is extremely toxic; if a cat accidentally ingests acetaminophen, seek immediate veterinary care)
- Steroids—dogs
- Griseofulvin (used to treat ringworm [known as “dermatophytosis”])—cats
- Halothane and methoxyflurane (gas anesthetic agents)—dogs
- Mebendazole—dogs
- Methimazole (used in the treatment of increased levels of thyroid hormone [known as “hyperthyroidism”])—cats
- Mitotane (Lysodren®, op’-DDD; used in the treatment of increased levels of steroids produced by the adrenal glands [known as “hyperadrenocorticism” or “Cushing’s disease”])—dogs
- Medications to control seizures, such as phenytoin, primidone, and phenobarbital—dogs
- Medication to improve appetite and weight gain (stanazolol)—cats
- Sulfa antibiotics—dogs (including trimethoprim-sulfadiazine)
- Tetracycline (an antibiotic)—dogs, cats
- Thiacetarsamide (medication to kill adult heartworms)—dogs, cats

Common Environmental Toxins

- *Amanita* mushrooms
- Aflatoxins/mycotoxins
- Blue-green algae (Cyanobacteria)
- Chlorinated compounds
- Cycad (sago palm nuts)
- Heavy metals (such as lead, zinc, copper)
- Phenols (especially cats)

Bacterial Toxins (known as “Endotoxins”)

- Intestinal bacteria—*Clostridium perfringens*; *Clostridium difficile*; gram-negative bacteria
- Food poisoning—*Staphylococcus*; *E. coli*; *Salmonella*

Nutritional / Herbal Products

- Lipoic acid—cats
- Kava Kava—dogs
- Comfrey extracts (pyrrolizidine alkaloids)
- Certain Chinese herbal medicines

RISK FACTORS

- Medications influencing liver function (such as phenobarbital, chloramphenicol, halothane, ranitidine, cimetidine, ketoconazole)
- Previous liver disease



TREATMENT

HEALTH CARE

- Inpatient—critical care setting required
- Prevention or correction of shock
- Fluid therapy—maintain liver blood flow, improve oxygen delivery and waste removal
- Colloid administration—colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume; plasma initially preferred for delivery of clotting substances
- Bleeding tendencies—provide vitamin K₁; administer fresh whole blood or fresh frozen plasma as needed (stored blood products may deliver high ammonia concentrations causing hepatic encephalopathy [brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia])
- Nasal oxygen—if patient has low blood pressure (known as “hypotension”) or fluid build-up in the lungs (known as “pulmonary edema”); may improve oxygen delivery to liver tissue

- Monitor urine output—initiate medications to remove excess fluids from the body (known as “diuretics”), as necessary
- Treat low blood sugar (hypoglycemia), if present—administer dextrose-containing solutions to maintain normal blood glucose levels

ACTIVITY

- Quiet and rest

DIET

- Protein—normal, unless overt hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)
- Nutrition via the gastrointestinal tract (as with a feeding tube)—small volume, frequent meals to optimize digestion and absorption of nutrients and to minimize formation of intestinal toxins that may contribute to hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)
- Nutrition through intravenous route (known as “parenteral nutrition”)—may be used as a partial means of providing nutrition (recommended for short-term lack of appetite) to minimize breakdown of muscle (catabolism) or as a total means of providing nutrition (known as “total parenteral nutrition” or “TPN”), which is recommended if the pet has lack of appetite of more than 5 days and providing nutrition via the gastrointestinal tract is not possible
- Ensure food intake



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Fluid therapy with judicious supplementation of potassium
- Short-acting steroids—for endotoxic shock (steroid such as prednisolone sodium succinate)
- Antibiotics—ampicillin, metronidazole, imipenem or ticarcillin (intravenous) with aminoglycoside or enrofloxacin—to protect against infections derived from migration of intestinal bacteria into the body
- Antioxidant therapy—crisis intervention: *N*-acetylcysteine for sudden (acute) or very sudden, very severe (known as “fulminant”) death of liver tissue (hepatic necrosis); when patient can accept oral medications and condition stabilizes change to *S*-adenosylmethionine (SAME, [Denosyl-SD4®]), which is a liver protectant against various hepatotoxins; Vitamin E (d-alpha-tocopherol acetate)
- B-complex vitamins
- Vitamin K₁—necessary for blood clotting
- Silibinin (milk thistle extract); benefits best characterized for *Amanita* mushroom toxicity; may improve liver regeneration; provides a variety of effects, such as antioxidant and liver protection
- Ursodeoxycholic acid—for long-term (chronic) liver disease (known as “hepatopathy”)
- Provide taurine supplementation to cats, especially in cats that are not eating; taurine is an amino acid (protein) that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the heart and retina (back of the eye)



FOLLOW-UP CARE

PATIENT MONITORING

- Prevent low body temperature (known as “hypothermia”)
- Monitor blood work (such as blood glucose, electrolytes, and packed cell volume [“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood])—monitor daily; fluctuations may occur rapidly
- Complete blood count (CBC), serum biochemical analysis, blood-clotting test(s)—repeat every 48 hours, as warranted

PREVENTIONS AND AVOIDANCE

- Close scrutiny of environment and future medications

POSSIBLE COMPLICATIONS

- Blood-clotting disorder (disseminated intravascular coagulopathy or DIC)
- Brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (hepatic

encephalopathy)

- Progressive liver failure
- Damage and scarring of the liver (known as “cirrhosis”)

EXPECTED COURSE AND PROGNOSIS

- Three to five days of treatment and observation are needed to estimate prognosis
- Progressive worsening of status: vomiting that does not respond to treatment and vomiting of blood (known as “hematemesis”), intolerance to supportive treatments, production of only small amounts of urine (known as “oliguria”), blood-clotting disorder (disseminated intravascular coagulopathy or DIC), and the brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (hepatic encephalopathy)—negative prognostic indicators
- Damage and scarring of the liver (cirrhosis)—possible in 3 to 6 months



KEY POINTS

- Potential for the need for 3 to 10 days in intensive care (ICU)
- Damage and scarring of the liver (cirrhosis) develops in some patients after widespread liver injury; some patients develop long-term (chronic) inflammation of the liver (known as “chronic hepatitis”); others recover completely

DISEASE CAUSED BY *HISTOPLASMA*, A TYPE OF FUNGUS (HISTOPLASMOSIS)



BASICS

OVERVIEW

- “Histoplasmosis” is a generalized (systemic) fungal infection caused by *Histoplasma capsulatum*

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats and dogs

Mean Age and Range

- Cats—predominantly young; many less than 1 year of age; all ages can be infected
- Dogs—most often young to middle-aged; all ages can be infected

SIGNS/OBSERVED CHANGES in the ANIMAL

Cats

- Subtle onset, over days to weeks
- Lack of appetite (known as “anorexia”), weight loss, and difficulty breathing (known as “dyspnea”)—most common
- Coughing occasionally
- Increased breathing effort and harsh lung sounds
- Lameness
- Eye discharges
- Diarrhea
- Fever to 40° C (104.0° F)
- Pale gums and moist tissues of the body (known as “mucous membranes”)
- Enlarged lymph nodes

Dogs

- Weight loss, depression, and diarrhea with straining—most common
- Coughing
- Difficulty breathing (dyspnea), associated with harsh lung sounds
- Exercise intolerance
- Enlarged lymph nodes (known as “lymphadenopathy”)
- Lameness and eye and skin changes—less common
- Thin to emaciated
- Fever to 40° C (104.0° F)
- Enlargement of the liver and spleen (known as “hepatosplenomegaly”)
- Pale gums and moist tissues of the body (mucous membranes)
- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)

CAUSES

- *Histoplasma capsulatum*, a type of soil-borne fungus

RISK FACTORS

- Bird roosts where the soil is enriched with bird or bat droppings are high-risk environments; old chicken coops and caves have been implicated
- Exposure to airborne dust contaminated with fungal spores coming from sites of fungal growth (especially a source of exposure for indoor cats)
- Tissue samples from nearly half of stray dogs and cats from areas where *Histoplasma* is common (known as “endemic areas”) were positive for *Histoplasma*, supporting the theory that many people and animals are exposed to the fungus and infected, but few develop clinically significant disease



TREATMENT

HEALTH CARE

- Usually outpatient with oral itraconazole (an antifungal drug)
- Inpatient with intravenous amphotericin B (an antifungal drug)—dogs with severe intestinal disease and abnormal absorption of food (known as “malabsorption”)
- Dogs on amphotericin B therapy—keep well hydrated with a balanced electrolyte solution to decrease potential for kidney toxicity (a side effect of amphotericin B)
- Emaciated animals with abnormal absorption of food (malabsorption)—provide nutrition through intravenous feedings to reverse wasting, until the intestinal disease is resolved enough for adequate food absorption
- Animals with severe difficulty breathing (dyspnea)—provide oxygen supplementation

ACTIVITY

- Dogs with difficulty breathing (dyspnea)—reduce activity

DIET

- Good-quality, easily absorbed, palatable food required



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Itraconazole

- Antifungal drug
- Drug of choice, if adequate intestinal function for drug absorption exists
- Dogs and cats—give with a high-fat meal
- Be cautious about compounded itraconazole because absorption of the drug may not be good
- Duration of treatment depends on clinical response; minimum treatment is 90 days

Amphotericin B

- Antifungal drug, must be administered intravenously

Dogs

- With severe inflammatory bowel disease and abnormal absorption of food (malabsorption)—use until patient begins to gain weight; then start on itraconazole
- Patient must be well hydrated before starting treatment
- Check blood work (blood urea nitrogen [BUN]) before each dose; discontinue treatment if BUN approaches 50 mg/dl and maintain hydration; resume when BUN is less than 30 mg/dl

Cats

- Use cautiously
- More sensitive to the drug than are dogs

Fluconazole

- Antifungal drug
- Eye and brain involvement may best be treated with fluconazole that penetrates the blood-brain barrier
- Use for dogs that cannot be given amphotericin B
- Intravenous form—administered until intestinal absorption allows oral itraconazole treatment



FOLLOW-UP CARE

PATIENT MONITORING

- Blood work (serum alanine aminotransferase [ALT])—with itraconazole treatment; check monthly or if the patient has a lack of appetite (anorexia)
- Chest X-rays—with lung involvement; check after 60 days of treatment to assess improvement; repeat at 30-day intervals and stop treatment when lungs are clear or remaining lung lesions fail to improve, indicating residual scarring; may be difficult to differentiate between residual scarring and active disease; continue treatment for at least 1 month after all signs of active disease have resolved
- Monitoring urinary *Histoplasma* antigen levels may be helpful; “antigens” are substances that induce an immune response by the body

PREVENTIONS AND AVOIDANCE

- Avoid suspected areas of exposure (such as bird roosts)
- Recovered dogs are probably immune

POSSIBLE COMPLICATIONS

- Recurrence possible; requires a second course of treatment

EXPECTED COURSE AND PROGNOSIS

- Treatment—duration is usually about 4 months; drugs are expensive, especially for large dogs
- Prognosis—good for stable patients without severe difficulty breathing (dyspnea); influenced by severity of lung involvement and physical weakness of the patient



KEY POINTS

- Discuss possible areas of exposure in the home environment with your pet’s veterinarian
- Both pets and family members may have been exposed to the same source of *Histoplasma*
- The animal is not a potential source of *Histoplasma* infection to the family; however, the environment is a source of possible infection for the animal and family members

HOUSESOILING—CATS: INAPPROPRIATE URINATION AND URINE MARKING



BASICS

OVERVIEW

- Urinating or marking territory in a location that the owner considers inappropriate
- Housesoiling includes inappropriate urination by indoor cats and urine marking
- Inappropriate urination is characterized by the cat simply squatting and urinating on horizontal surfaces outside of the litter box
- Urine marking occurs most commonly when the cat sprays urine on vertical surfaces outside the litter box; also known as “urine spraying”
- The “lower urinary tract” includes the urinary bladder and the urethra (the tube from the bladder to the outside, through which urine flows out of the body)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Predominant Sex

- Urine marking or spraying is more common in intact and neutered males than in females

SIGNS/OBSERVED CHANGES in the ANIMAL

Inappropriate Urination

- Sudden (acute) or long-term (chronic) problem
- Signs of lower urinary tract disease (such as straining to urinate) or generalized (systemic) illness may suggest an underlying medical problem.
- Presence of abnormal physical findings depends on whether problem is a disease-related or behavioral problem

Urine Marking

- Usually manifest as spraying—the cat moves so the rear quarters are close to a vertical surface, the cat stiffens its posture, raises and quivers its tail, and directs a small burst of urine toward the vertical surface
- Urine marks may be detected around doorways or windows or prominent furniture or on new objects brought into the house
- Horizontal urine marks may be found on clothing or bedding associated with a particular person or on new objects brought into the house

CAUSES

Medical Abnormalities Associated with Inappropriate Urination

- Lower urinary tract disease (such as feline lower urinary tract disease [FLUTD] or lower urinary tract infection)
- Presence of stones (known as “uroliths”) in the urinary tract (condition known as “urolithiasis”)
- Diabetes mellitus (“sugar diabetes”)
- Excessive levels of thyroid hormone (known as “hyperthyroidism”)
- Feline leukemia virus (FeLV) infection
- Feline immunodeficiency virus (FIV) infection
- Liver disease
- Senility or decline in thinking, learning, and memory, frequently associated with aging (known as “cognitive dysfunction”)
- Caused by or related to medical treatment (known as an “iatrogenic abnormality”)—administration of fluids, steroids, medications to remove excess fluids from the body (known as “diuretics”)

Environmental Factors Contributing to Urine Housesoiling

Litter Box Characteristics

- Soiled litter box
- Inadequate number of litter boxes or locations (one litter box per cat plus one is recommended)
- Litter box located in remote or unpleasant surrounding or subject to interference by dogs or children
- Inappropriate type of litter box—a covered litter box may maintain odors at an offensive level or may be too small to allow large cats

to move around comfortably; a covered litter box allows other cats, pet dogs, and young children to “target” the cat as it exits

- Time factors—daily or weekly patterns of inappropriate urination suggest an environmental cause; sudden (acute) onset in a cat that previously has used the litter box reliably suggests a medical problem
- Type of litter—litter type that is unacceptable to the cat (for example, scented litter may not be acceptable); preference tests indicate that most cats prefer unscented, fine-grained (clumping) litter; change in litter-box habits that coincide with introduction of a new litter type suggests an association with the change of litter
- Sudden shift from using litter in the litter box to urinating in an unusual location (such as urinating in a porcelain sink) suggests a lower urinary tract disorder
- Location—urination outside the litter box may suggest a location preference or influential social factors
- Social dynamics—consider social conflicts between cats and any changes in the social world of the cat at the time the problem started (such as addition of a new cat to the household)

Environmental Factors Contributing to Urine Marking

- Probability of urine marking or spraying is directly proportional to the number of cats in the household
- Presence of outdoor cats may elicit urine marking or spraying around doorways and windows
- Urine marking or spraying may be a response to another cat in the home or outside the home
- Urine marking or spraying on grocery bags or new furniture suggests olfactory marking, associated with arousal in response to new stimuli
- Urine marking or spraying on clothing or bedding may be associated with specific people or visitors

RISK FACTORS

Inappropriate Urination

- Infrequently changed/cleaned litter box (or boxes)
- Frequent travel by owner (possible sign of separation anxiety in the cat)

Urine Marking

- History of urine marking by a parent of the cat
- Multiple-cat households



TREATMENT

HEALTH CARE

- Treat any underlying medical condition
- Use environmental and behavioral therapies before or with medical treatment (See www.vet.osu.edu/indoorcat)
- Use barriers or electronic motion detectors to restrict the cat from rooms in which urine housesoiling occurs
- If the owner requires immediate cessation of the problem, it is helpful to confine the cat to one room in the owner's absence; provide a litter box, water, food, and resting sites in this room; the cat can be let out of the room when the owner returns and is available for strict supervision of the cat
- Clean urine “accidents” with an enzymatic cleaner specific for this purpose
- Number the litter boxes and count and record the number of urinations in each box and outside the litter boxes each day

Inappropriate Urination

Environmental Management Techniques

- Scoop out the litter boxes daily and clean thoroughly weekly and refill
- Avoid deodorizers or other strong odors in the vicinity of the litter box
- Move food bowls away from the litter box
- Provide at least one litter box per cat, distributed in more than one location, and avoid high traffic or noisy areas
- If the litter box is covered, provide an additional large, plain, uncovered litter box filled with unscented, fine-grained, clumping litter, with no liner
- Additional boxes may be provided, using a different type of litter in each (so called “litter-box buffet”) to evaluate the cat's preference for litter-box type and litter
- If one site in the home is preferred for inappropriate urination, place another litter box over this site—after the cat uses this box regularly, move it gradually (several inches a day) to a site more acceptable to the owner
- Confinement of the cat in a “safe room” when the owner is not available to supervise may be necessary

Behavior Modification

- Punishment (such as using a water pistol or sound alarm) is not effective and will increase the cat's anxiety
- Punishment associated with sounds or movements by the owner will condition the cat to avoid the owner
- Feeding or playing with the cat at the location(s) where the cat is urinating inappropriately may countercondition the unacceptable behavior; "counterconditioning" is training the cat to demonstrate a positive behavior in place of the negative behavior (in this case, inappropriate urination)

Urine Marking

- Neuter intact male cats and spay intact female cats—this curbs spraying behavior in up to 90% of males and 95% of females that spray
- If signs suggest that the cat is spraying in response to cats outside their house, prevent visual or olfactory access to those cats; an environmental product (Feliway®, Veterinary Product Laboratories), a concentrate of synthesized feline facial pheromone, is available commercially as a treatment for urine marking—the product is sprayed regularly or diffused in the environment and may improve urine spraying in up to 75% of cases
- Block "inside" cat's ability to see "outside" cats
- Spend time interacting with the cat daily to focus the affected cat's attention away from other cats
- Medications play an important role in the control of urine marking



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Inappropriate Urination

- Usually are not indicated, except in treatment-resistant cases or when inappropriate urination is associated with generalized anxiety

Urine Marking

- Medications to decrease arousal and anxiety; medications commonly used include clomipramine, amitriptyline, buspirone, fluoxetine

Other Medications

- Synthetic progestins—the risk of serious side effects has diminished their once-common use; discuss the risks and benefits with your cat's veterinarian



FOLLOW-UP CARE

PATIENT MONITORING

- Keep a daily log of urination patterns so that treatment success can be evaluated and appropriate adjustments in treatment can be made
- Regular follow-up is essential

POSSIBLE COMPLICATIONS

- Treatment failure may result in the cat being euthanized, relinquished at an animal shelter, or released outside

EXPECTED COURSE AND PROGNOSIS

- Client expectations must be realistic—immediate control of a longstanding problem of housesoiling is unlikely; the goal is gradual improvement over time



KEY POINTS

- Urinating or marking territory in a location that the owner considers inappropriate
- Inappropriate urination is characterized by the cat simply squatting and urinating on horizontal surfaces outside of the litter box
- Urine marking occurs most commonly when the cat sprays urine on vertical surfaces outside the litter box; also known as "urine spraying"
- Client expectations must be realistic—immediate control of a longstanding problem of housesoiling is unlikely; the goal is gradual improvement over time

HOUSESOILING—DOGS



BASICS

OVERVIEW

- Urinating and/or defecating (having a bowel movement), as a means of eliminating or marking territory, in a location that the owner considers inappropriate

GENETICS

- Some dog breeds appear to be housetrained more easily than other dog breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Potential genetic breed-related likelihood for ease of housetraining and submissive or excitement urination (“submissive urination” occurs when someone enters the room or home and the dog urinates to signal that s/he is insubordinate to the person; this is “normal” dog-greeting behavior and is seen especially in dogs that are shy or do not have self confidence—unfortunately, this “normal” dog behavior is not desirable; “excitement urination” occurs when a dog gets overly excited or enthusiastic and it leaves “dribbles” of urine at your feet)

Mean Age and Range

- Inappropriate elimination due to improper housetraining primarily seen in younger dogs
- Submissive and excitement urination seen primarily in younger dogs
- Urine marking begins to be displayed as the dog begins to reach sexual maturity

Predominant Sex

- Female dogs generally are easier to housetrain than male dogs
- Intact male dogs are more likely to urine-mark than neutered male dogs and intact or spayed female dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Urinating and/or defecating in inappropriate areas (according to the owners), usually inside a home
- Abnormal physical examination findings would be related to an underlying medical cause of inappropriate elimination

CAUSES

- Causes of housesoiling can be related to a primary behavioral problem or secondary to or in association with a medical disorder
- May be associated with signs of other behavioral disorders (such as separation anxiety)
- May be associated with lack of time spent on owner’s part to teach housetraining properly
- May be associated with punishment of a dog that submissively urinates, which may make the problem worse
- Determine potential triggers, via a complete behavioral history, including when, where, and how often the elimination occurs and reliability of outdoor elimination
- If no abnormal physical examination findings are identified, the housesoiling is probably due to a behavioral cause

Behavioral Causes

- Lack of or incomplete housetraining
- Marking behavior
- Submissive urination
- Excitement urination
- Separation anxiety syndrome
- Cognitive dysfunction syndrome (condition in which older dog is confused, forgetful, and may lose its housetraining)
- Noise phobia
- Fear-induced
- Excessive thirst due to psychological need to drink water (known as “psychogenic polydipsia”)

Medical Causes

Degenerative Abnormalities

- Hip dysplasia/osteoarthritis/degenerative joint disease

- Kidney failure

Anatomic or Structural Abnormalities

- Ectopic ureters (condition in which one or both ureters [tube from the kidney to the bladder] insert into the bladder in an unusual location, frequently leading to dribbling of urine)

Metabolic Disease

- Incontinence (inability to control urination and/or defecation)
- Diabetes mellitus (“sugar diabetes”)
- Diabetes insipidus (“water diabetes”)
- Liver insufficiency
- Excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Inadequate production of steroids by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”)

Cancer

- Kidney cancer
- Bladder cancer
- Other cancers causing weakness

Infectious/Inflammatory Diseases

- Urinary tract infection
- Crystals in the urine (known as “crystalluria”) in association with bladder inflammation (known as “cystitis”) or stones in the urinary tract (known as “urolithiasis”)

RISK FACTORS

- Intact male
- Owners poorly informed or motivated to properly houstrain their dog



TREATMENT

HEALTH CARE

- Any appropriate measures to assure continued good health of the dog

ACTIVITY

- Take dog outside often to ensure that s/he has enough access to eliminate outside, or provide acceptable access to the outside, for example via a dog door
- Increase activity level to help in the treatment of separation anxiety, as well as to improve the dog’s health

DIET

- If the dog is eliminating bowel movement inappropriately, feeding meals at specific times (as opposed to free-choice feeding) may help in maintaining the dog on a schedule of defecation
- Feeding a diet of higher caloric density may help decrease the urge to defecate as often

SURGERY

- Neutering an intact male dog decreases urine marking rapidly in 30% of dogs, with a gradual decline in 20% of dogs, and no change in 50% of male dogs; the results are the same regardless of the age of neutering



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- If the dog is urine marking or inappropriately eliminating owing to anxiety, medications may be helpful, but only in conjunction with behavior modification
- Selective serotonin reuptake inhibitors (SSRIs) or tricyclic antidepressants/anti-anxiety medications (TCAs) may be helpful; an

example of a SSRI is fluoxetine; an example of a TCA is clomipramine

- Full onset of action of these medications can be four-to-six weeks after initiation of treatment, and owners need to understand the amount of time necessary before seeing response
- Side effects of TCAs can include nausea; vomiting; diarrhea; sluggishness (lethargy); irregular heart beats (known as “cardiac arrhythmias”); and enhancement of seizure activity
- Side effects of SSRIs can include nausea; vomiting; diarrhea; and sluggishness (lethargy)
- Drugs are much less effective if anxiety is not part of the problem; will have negligible effect in dogs that are not housetrained or in dogs with submissive urination
- Progestins were used in the past to control urine marking, but are rarely recommended because of potential severe side effects



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor progress with the owner through follow-up visits or telephone calls; the owner should keep a journal of incidents, inciting factors, and treatments instituted to give an objective view of improvement

PREVENTIONS AND AVOIDANCE

- Properly housetrain the dog
- Neuter male dogs and spay female dogs
- Treat any underlying behavioral condition
- Treat any underlying medical condition

POSSIBLE COMPLICATIONS

- Inappropriate elimination is the most common individual reason for relinquishment of a pet to a shelter
- Recurrence of housesoiling may happen, if owner does not continue medical and behavioral treatment

EXPECTED COURSE AND PROGNOSIS

- Prognosis for any behavioral problem is highly dependent on the owner’s ability to fully follow instructions; rarely are dogs with behavioral problems considered “cured,” but instead, they are “managed”
- The following estimations of prognosis are based on the owner following your instructions for behavior modification:
 - Prognosis for decreasing submissive and excitement urination is good
 - Prognosis for managing incomplete housetraining is good
 - Prognosis for urine marking in previously intact male: 50% improve (30% quickly, 20% more slowly) with neutering, even without behavior modification
 - Prognosis for managing urine marking in spayed or neutered dogs is good, if the triggers can be identified and managed with avoidance or other forms of behavior modification
- Some animals with underlying medical causes of inappropriate elimination can still eliminate inappropriately after the medical cause has been treated properly



KEY POINTS

- Proper housetraining should be stressed with clients from the very beginning
- Potential long-term management necessary to control the problem
- Treat underlying/contributing medical problems
- Treat other underlying/contributing behavioral problems
- Clean the soiled areas with an enzymatic cleaner, to help eliminate any odor that may attract the dog to eliminate in that location again; if the object soiled is a piece of clothing, wash it in the washing machine
- Inappropriate elimination is the most common individual reason for relinquishment of a pet to a shelter

Incomplete Housetraining

- Keep the dog completely supervised or confined at all times
- Take the dog outside frequently to eliminate

- Reward the dog for eliminating at the appropriate time and place; requires the owner to go outside with the pet
- Thoroughly clean soiled areas

Submissive Urination

- Do not punish the behavior, since this may make problem worse
- Ignore the dog when s/he comes into the house (no verbal or physical interactions or eye contact)
- The dog should go outside to eliminate before being greeted by any person, including family members
- The dog should be greeted in a non-confrontational and quiet manner; do not lean over the dog or institute interactive play at the time of greeting
- Alternative activities at homecoming (such as asking for a toy or requesting a “sit”) may help in mild cases
- For excitement urination, much the same recommendations are applicable as for submissive urination, especially concentrating on not getting the dog overexcited

Urine-Marking Behavior

- Determine any possible triggers to the behavior, including anxiety-provoking stimuli
- Address those triggers with desensitization and counterconditioning and/or avoidance of the trigger, as appropriate
- Neutering is effective in many dogs to decrease urine marking
- Make the areas urine marked aversive to the dog by use of “booby traps,” such as upside-down plastic carpet runners or mousetraps, or by use of remote punishment at the very beginning of each and every urine-marking episode, if the owner is able to catch the dog in the act of marking
- Prevent access to preferred marking locations
- Alternatively, change the significance of the area to a positive place, by feeding the dog in the area marked

“WATER ON THE BRAIN” (HYDROCEPHALUS)



BASICS

OVERVIEW

- The cerebrum is the large rounded structure of the brain; it is composed of two hemispheres; inside each hemisphere is a cavity, known as a “lateral ventricle;” the two lateral ventricles and two other ventricles (third and fourth ventricles) form the “ventricular system” of the brain; cerebrospinal fluid is produced from specialized areas in the ventricles; the cerebrospinal fluid flows between the ventricles and into the spinal canal; blockage of flow can lead to increased pressure in the lateral ventricles, resulting in hydrocephalus
- Cerebrospinal fluid (also known as “CSF”) is a specialized body fluid that cushions the brain and spinal cord
- Hydrocephalus is a condition in which the ventricular system of the brain is abnormally dilated from an increased volume of cerebrospinal fluid
- May be symmetrical or asymmetrical
- May involve the entire ventricular system or only parts of the ventricular system if a blockage of the flow of cerebrospinal fluid is present, in which case, the CSF volume increases in the part of the ventricular system behind the blockage (in other words, the blockage acts as a dam to the flow of CSF)
- Lay term for hydrocephalus is “water on the brain”

GENETICS

- Inherited hydrocephalus—Siamese cats and Yorkshire terrier
- Siamese cats—autosomal recessive

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Congenital (present at birth) hydrocephalus—small and short-nosed, flat-faced (known as “brachycephalic”) dogs: bulldogs, Chihuahuas, Maltese, Pomeranians, toy poodles, Yorkshire terriers, Lhasa apsos, cairn terriers, Boston terriers, pugs, and Pekingese
- Inherited hydrocephalus—Siamese cats and Yorkshire terrier; high incidence of enlargement of the ventricles (known as “ventriculomegaly”) that do not cause clinical signs in normal adult beagles
- Acquired (condition that develops sometime later in life/after birth) hydrocephalus—any breed of dog or cat

Mean Age and Range

- Congenital (present at birth) hydrocephalus—usually becomes apparent at a few weeks up to 1 year of age; sudden (acute) onset of signs can occur in dogs with previously undiagnosed congenital hydrocephalus—the exact cause of this sudden onset of signs is uncertain
- Acquired (condition that develops sometime later in life/after birth) hydrocephalus—any age

Predominant Sex

- None

SIGNS/OBSERVED CHANGES in the ANIMAL

- Congenital (present at birth) hydrocephalus—may occur without clinical signs, especially in dogs of toy breeds; other malformations or abnormalities of the central nervous system may be present, which may contribute to clinical signs
- Acquired (condition that develops sometime later in life/after birth) hydrocephalus—signs from the underlying disease (that is, the disease that led to the development of hydrocephalus) may be as or more prominent than signs from hydrocephalus itself
- Behavioral signs—decreased awareness; lack of or loss of training ability (including house-training); excessive sleepiness; vocalization; sometimes hyperexcitability
- Blindness
- Seizures—may be noted
- Head—may appear large and dome-shaped; persistent “soft spot” of the head
- Outward and downward deviation of both eyes (known as “bilaterally divergent strabismus”) is present in some dogs with severe congenital (present at birth) hydrocephalus
- Gait abnormalities—incoordination, wobbly or “drunken” gait (known as “ataxia”)
- Severely increased pressure within the head (known as “intracranial pressure”)—stupor or coma, pinpoint or dilated fixed pupils, and abnormal breathing patterns; may lead to fatal condition in which the brain has pushed downward in the skull and has herniated through the opening that leads to the neck (known as “brain herniation” or “tentorial herniation”)

CAUSES

- Congenital (present at birth) hydrocephalus—inherited malformation of the ventricular system; prenatal infection (dogs—parainfluenza virus; cats—coronavirus); exposure to compounds that cause abnormal development of the embryo (known as “teratogenic compounds”); bleeding into the brain of the fetus or newborn secondary to a difficult birth (known as “dystocia”); nutritional deficiency (vitamin A)
- Acquired (condition that develops sometime later in life/after birth) hydrocephalus—inflammatory diseases or mass lesions within the skull

RISK FACTORS

- Animals with hydrocephalus that do not have clinical signs may develop clinical signs in the face of an insult, such as infection or trauma



TREATMENT

HEALTH CARE

- Inpatient—intensive care for patients with severe signs or when undergoing surgical therapy
- Outpatient—patients with mild to moderate signs that can be treated medically
- Prevent secondary complications of lying down for prolonged periods in stuporous or comatose patients—avoid pressure sores; drying eyes; and lung congestion

ACTIVITY

- Depends on severity of signs

SURGERY

- Surgical procedure in which cerebrospinal fluid is shunted from the ventricles of the brain to the abdominal cavity or to the right atrium of the heart—definitive treatment; clinical signs may not resolve completely; residual signs usually indicate irreversible brain damage
- Surgery for a brain tumor or other mass lesion—consider if it is the underlying cause of hydrocephalus



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Reduce production of cerebrospinal fluid—steroids (such as prednisone or dexamethasone); or carbonic anhydrase inhibitors (such as acetazolamide); omeprazole has been reported to reduce CSF production in the dog in an experimental model, but no data is available on the usefulness of this drug in treating hydrocephalus
- Reduce increased pressure within the head (intracranial pressure)—medications to decrease excess fluid in the body (known as “diuretics,” such as mannitol and/or furosemide); these are short-term treatments only, helpful for immediate treatment of severe cases
- Treat underlying cause—administer specific drugs when possible (such as antibiotics for bacterial infection)



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor for increase in severity of signs of hydrocephalus and for signs from an underlying cause (such as a tumor in the skull)

POSSIBLE COMPLICATIONS

- Infection and shunt blockage can occur following surgical shunting of cerebrospinal fluid; shunt revision commonly is needed
- Brain pushes downward in the skull and herniates through the opening that leads to the neck (brain or tentorial herniation) and death

EXPECTED COURSE AND PROGNOSIS

- Good to poor: depends on cause and severity of signs of hydrocephalus
- Mild congenital (present at birth) hydrocephalus—good prognosis; may require only occasional medical treatment



KEY POINTS

- Observe for deterioration in mental alertness, vision, and behavior, which may signal worsening of hydrocephalus
- Severity of the clinical signs may not correspond to the degree of ventricular enlargement of the brain

HYPERADRENOCORTICISM OR CUSHING'S DISEASE IN DOGS

(EXCESSIVE LEVELS OF STEROIDS IN THE BODY)



BASICS

OVERVIEW

- “Spontaneous” hyperadrenocorticism (“HAC”) or Cushing’s disease is a disorder caused by excessive production of steroids by the adrenal glands; “spontaneous” denotes lack of apparent cause
- Spontaneous hyperadrenocorticism is a hormonal disorder; it can involve problems in the pituitary gland (the “master gland” of the body), in which case the pituitary gland “directs” the adrenal glands to produce excessive amounts of steroids—this type is known as “pituitary-dependent hyperadrenocorticism” or “PDH” or it can involve problems in the adrenal gland itself (benign tumors or cancer), in which the adrenal glands produce excessive amounts of steroids on “their own” and not under the control of the pituitary gland
- “Iatrogenic” hyperadrenocorticism or Cushing’s disease results from the use of medications containing steroids; the medications can be given by mouth or by injection, or can be applied topically to the skin, ears, or eyes; the signs of iatrogenic Cushing’s disease usually are related to the dose of steroids and length of treatment, but some dogs are very sensitive to steroids and may show signs with normal doses of steroids or relatively short length of treatment; “iatrogenic” refers to changes induced by the medication itself, the changes generally are unfavorable
- In both types of hyperadrenocorticism, clinical signs are due to the deleterious effects of the elevated circulating steroid concentrations on multiple organ systems

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Poodles, dachshunds, Boston terriers, boxers, and beagles reportedly are at increased risk as compared to other breeds

Predominant Sex

- No increased likelihood for either sex has been identified for pituitary-dependent hyperadrenocorticism (PDH) in dogs; possible increased likelihood that female dogs will have adrenal tumors that produce excessive steroids

Mean Age and Range

- Hyperadrenocorticism (HAC) is generally a disorder of middle-aged to old animals; pituitary-dependent hyperadrenocorticism (PDH) very rarely can be seen in dogs as young as 1 year of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Severity varies greatly, depending on the duration and severity of steroid excess
- In some cases, the physical presence of cancer in the pituitary gland (leading to pituitary-dependent hyperadrenocorticism [PDH]) or the adrenal gland itself contributes to signs
- Excessive urination (known as “polyuria”) and increased thirst (known as “polydipsia”); increased appetite (known as “polyphagia”); sagging abdomen due to weakened abdominal muscles (known as “pendulous abdomen”); enlarged liver (known as “hepatomegaly”); hair loss; sluggishness (lethargy); muscle weakness; lack of heat cycles in female dogs (known as “anestrus”); obesity; loss of muscle mass (known as “muscle atrophy”); “blackheads” (known as “comedones”) on the skin; increased panting; loss of size or tissue of the testicles (known as “testicular atrophy”); darkening of the skin (known as “hyperpigmentation”); calcium deposits in the skin (known as “calcinosis cutis”); facial nerve paralysis; thin skin; bruising

CAUSES

- Pituitary-dependent hyperadrenocorticism (PDH)—benign tumor (known as a “pituitary adenoma”) most common; cancer (known as a “pituitary adenocarcinoma”) rare
- Adrenal gland itself (not under control of pituitary gland)—benign tumor (known as “adrenal adenoma”) or cancer (known as “adrenal carcinoma”)
- Iatrogenic hyperadrenocorticism—due to administration of steroid-containing medications

RISK FACTORS

- Poodles, dachshunds, Boston terriers, boxers, and beagles reportedly are at increased risk, as compared to other breeds



TREATMENT

HEALTH CARE

- Dictated by the severity of clinical signs, the patient's overall condition, and any complicating factors (such as diabetes mellitus ["sugar diabetes"], blood clots to the lungs [known as "pulmonary thromboembolism"])

ACTIVITY

- No alteration of activity necessary

DIET

- Usually no need to alter diet; use appropriate diet if dog also has diabetes mellitus (sugar diabetes)

SURGERY

- Surgical removal of the pituitary gland (known as "hypophysectomy")—described in veterinary literature, but generally not available
- Surgical removal of both adrenal glands (known as "bilateral adrenalectomy") not used for treatment of pituitary-dependent hyperadrenocorticism (PDH) in dogs
- Surgery is the treatment of choice in dogs with adrenal adenomas (benign tumors of the adrenal gland) and small carcinomas (cancer of the adrenal gland), unless the patient is a poor surgical risk or the client refuses surgery; appropriate personnel and facilities are required as this is a technically demanding surgery and intensive postoperative management is required
- Depending on patient status, medical control of hyperadrenocorticism (HAC) may be desirable prior to surgery, if possible



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Mitotane

- Mitotane (also known as "*o,p'*-DDD" or "Lysodren®") remains the drug of choice for medical management of both pituitary-dependent hyperadrenocorticism (PDH) and adrenal tumors in dogs; it selectively destroys the steroid-secreting cells of the adrenal gland
- Inadequate levels of aldosterone (another hormone produced by the adrenal gland; involved in sodium and potassium regulation in the body) are possible secondary to mitotane therapy; if occurs, likely patient will have permanent complete adrenal-gland insufficiency; treatment for inadequate or too low levels of steroids produced by the adrenal glands (known as "hypoadrenocorticism" or "Addison's disease") should be initiated

***l*-Deprenyl**

- *l*-Deprenyl (also known as "selegiline hydrochloride" or Anipryl®) may be used as an alternative treatment for pituitary-dependent hyperadrenocorticism (PDH); it decreases secretion of the pituitary hormone (known as "ACTH") that directs the adrenal gland to produce steroids, thus decreasing serum steroid concentrations; not recommended for treatment of PDH in dogs with concurrent illnesses, such as diabetes mellitus
- Effectiveness of the drug in treating PDH is questionable; one study found 20% efficacy and another found it to be ineffective
- Adverse effects (such as lack of appetite [anorexia], sluggishness [lethargy], vomiting and diarrhea) are uncommon and usually mild; disadvantages include the need for lifelong daily administration and the expense of the medication

Ketoconazole

- Ketoconazole inhibits enzymes responsible for steroid production; indicated for dogs unable to tolerate mitotane at doses necessary to control hyperadrenocorticism (HAC); may be useful for control of clinical signs in dogs with adrenal tumors; effective in approximately 50% or fewer cases; adverse effects include lack of appetite (anorexia), vomiting, diarrhea, sluggishness (lethargy), and liver disease

Trilostane

- Trilostane (Vetoryl®) is approved for use in Europe and available in the United States by special application to the federal Food and Drug Administration (FDA) for compassionate use license—your pet's veterinarian will need to contact the FDA; efficacy for treatment of pituitary-dependent hyperadrenocorticism (PDH) is high, comparable to mitotane; survival of dogs with PDH is the same for dogs treated with mitotane or trilostane
- Trilostane can suppress aldosterone (another hormone produced by the adrenal gland; involved in sodium and potassium regulation in

the body) secretion, causing a sudden change in sodium and potassium regulation (known as an “Addisonian crisis”), which requires immediate medical treatment; fortunately, the low levels of aldosterone generally resolve within 48 to 72 hours of discontinuation of drug administration, but low levels of weeks’ to months’ duration has been reported

- Can be used to treat adrenal tumors and will control clinical signs, at least temporarily, but is not the drug of choice; for adrenal tumors, mitotane is the drug of choice, as it is truly chemotherapeutic and may kill tumor cells

Other Treatment—Radiation Therapy

- Consider radiation therapy for animals with large, benign pituitary tumors (known as “pituitary macroadenomas”)
- Decreases in secretion of the pituitary hormone (known as “ACTH”) that directs the adrenal gland to produce steroids, thus decreasing serum steroid concentrations levels, may take several months; control signs of hyperadrenocorticism (HAC) with previously described drugs in the interim



FOLLOW-UP CARE

PATIENT MONITORING

- Response to therapy
- Periodic blood work (such as complete blood count [CBC] and serum biochemistry profile as well as specific tests for adrenal gland function)

EXPECTED COURSE AND PROGNOSIS

- Untreated hyperadrenocorticism (HAC)—generally a progressive disorder, with a poor prognosis
- Treated pituitary-dependent hyperadrenocorticism (PDH)—usually a good prognosis; median survival time for a dog with PDH treated with mitotane or trilostane is approximately 1.9 years; at least 10% survive 4 years; dogs living longer than 6 months tend to die of causes unrelated to HAC
- Dogs with large, benign pituitary tumors (macroadenomas) and nervous system signs—poor to grave prognosis; dogs with macroadenomas with no or mild nervous system signs—fair to good prognosis with radiation and medical therapy
- Dogs with benign adrenal-gland tumors (adrenal adenomas)—usually a good to excellent prognosis; dogs with small adrenal-gland cancers (carcinomas) that have not metastasized have a fair to good prognosis.
- Dogs with large adrenal-gland cancers (carcinomas) and/or with widespread spread (metastasis)—generally a poor to fair prognosis, but impressive responses to high doses of mitotane are seen occasionally



KEY POINTS

- If using medical therapy, lifelong therapy required
- If adverse reaction to mitotane or trilostane occurs—discontinue drug, give prednisone, and have veterinarian reevaluate next day; if no response to prednisone is noted within a few hours of administration, veterinarian should evaluate immediately

HYPERCALCEMIA (HIGH LEVELS OF CALCIUM IN THE BLOOD)



BASICS

OVERVIEW

- Excessive or high levels of calcium in the blood (known as “hypercalcemia”)
- Serum total calcium greater than 11.5 mg/dL on blood work in dogs
- Serum total calcium greater than 10.5 mg/dL on blood work in cats

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog and cat

Breed Predilections

- Primary hyperparathyroidism in the keeshond (dog) and Siamese (cat); “hyperparathyroidism” is an abnormal condition in which high levels of parathyroid hormone are circulating in the blood; parathyroid hormone regulates calcium levels in the body—it increases calcium levels by causing calcium to be reabsorbed from bone; “primary hyperparathyroidism” refers to a condition in which a tumor in the parathyroid gland produces excessive levels of parathyroid hormone, leading to increased blood calcium levels

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on the cause of the high levels of calcium in the blood (hypercalcemia); many animals have no clinical signs
- Patients with underlying cancer, kidney failure, or low levels of steroids produced by the adrenal glands (known as “hypoadrenocorticism”) generally appear ill
- Patients with primary hyperparathyroidism (a condition in which a tumor in the parathyroid gland produces excessive levels of parathyroid hormone, leading to increased blood calcium levels) show mild clinical signs, if any, due solely to the effects of the high levels of calcium in the blood (hypercalcemia)
- Signs become apparent when high levels of calcium in the blood (hypercalcemia) are serious and long-term (chronic)
- Excessive urination (known as “polyuria” or “PU”) and excessive thirst (known as “polydipsia” or “PD”)—most common in dogs
- Lack of appetite (known as “anorexia”)
- Sluggishness (lethargy)—most common in cats
- Vomiting
- Constipation
- Weakness
- Impaired consciousness in which animal must be stimulated to be awakened (known as “stupor”) and unconsciousness in which animal cannot be stimulated to be awakened (known as “coma”)—severe cases
- Lower urinary tract signs in animals with secondary calcium-containing stones (known as “uroliths”); the lower urinary tract includes the bladder and urethra (the tube from the bladder to the outside, through which urine flows out of the body); lower urinary tract signs include straining to urinate and painful urination
- Enlarged lymph nodes (known as “lymphadenopathy”) or enlargement of abdominal organs (known as “abdominal organomegaly”) may be seen in patients with lymphoma (a type of cancer)
- Parathyroid gland benign tumors (known as “parathyroid gland adenomas”)—cannot be felt in dogs during physical examination by the veterinarian; often can be felt in cats with primary hyperparathyroidism (a condition in which a tumor in the parathyroid gland produces excessive levels of parathyroid hormone, leading to increased blood calcium levels), but can be confused with the thyroid gland (the parathyroid glands are very small glands that are located immediately adjacent to the thyroid gland in the animal’s neck)

CAUSES

- Cancer—lymphoma (most common in dogs, less common in cats); anal-sac apocrine-gland adenocarcinoma (dogs); multiple myeloma; lymphocytic leukemia; metastatic bone cancer; fibrosarcoma (cats); various types of carcinoma
- Primary hyperparathyroidism (a condition in which a tumor in the parathyroid gland produces excessive levels of parathyroid hormone, leading to increased blood calcium levels)
- Kidney failure—sudden (acute) or long-term (chronic)

- Inadequate production of steroids by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”)
- Vitamin D-rodenticide poisoning—vitamin D-rodenticides are designed to kill rodents (such as mice and rats); no longer marketed in the United States
- Vitamin D poisoning from plant or food sources
- Diseases that soften or destroy bone (known as “osteolytic diseases”)
- Aluminum toxicity
- High blood-calcium levels for unknown reasons (known as “idiopathic hypercalcemia”) in cats

RISK FACTORS

- Keeshond (dog) and Siamese (cat)—primary hyperparathyroidism (a condition in which a tumor in the parathyroid gland produces excessive levels of parathyroid hormone, leading to increased blood calcium levels)
- Kidney failure
- Cancer
- Use of calcium supplements or calcium-containing intestinal phosphate binders
- Use of calcitriol or other vitamin D preparations



TREATMENT

HEALTH CARE

- Inpatient care, because of the deleterious effects of high levels of calcium in the blood (hypercalcemia) and the need for fluid therapy
- Very high levels of calcium in the blood (hypercalcemia) are a medical emergency

ACTIVITY

- Depends on signs and underlying cause of high levels of calcium in the blood (hypercalcemia)

DIET

- Depends on underlying cause of high levels of calcium in the blood (hypercalcemia)

SURGERY

- Surgery may be indicated, based on cause of high levels of calcium in the blood (hypercalcemia), such as for cases with cancer as the underlying cause



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Normal saline—fluid of choice
- Avoid calcium-containing fluids
- Drugs to eliminate fluids from the body (known as “diuretics,” such as furosemide) and steroids can be useful in treatment
- Sodium bicarbonate may be useful, in combination with other treatments
- Mithramycin has been used in cases with very serious signs related to high levels of calcium in the blood (known as “hypercalcemic crises”); avoid its use if possible, because of associated toxic effects to the kidney (known as “nephrotoxicity”) and liver (known as “hepatotoxicity”)
- Calcitonin may be useful in the treatment of cases with high levels of vitamin D (known as “hypervitaminosis D”)
- Pamidronate has been used successfully for treatment of high levels of calcium in the blood (hypercalcemia) of various causes in dogs and cats



FOLLOW-UP CARE

PATIENT MONITORING

- Check blood work for serum calcium levels every 12 hours
- Kidney function tests and urinalysis—the first sign of damage to the kidney tubules may be casts (accumulations of cellular debris and other material in the shape of a kidney tubule) in the urine sediment of the urinalysis
- Monitor urine output, particularly if kidney failure characterized by inadequate urine production (known as “oliguric renal failure”) is suspected, in which case urine output should be measured carefully
- Hydration status must be monitored; indicators of “overhydration” (that is, too much fluid in the body) include increased body weight and fluid build-up in the lungs (known as “pulmonary edema”) or under the skin (known as “subcutaneous edema”)

POSSIBLE COMPLICATIONS

- High levels of calcium are toxic to the kidney tubules and can cause excessive urination (polyuria) and excessive thirst (polydipsia) and irreversible kidney failure; can lead to formation of stones in the urinary tract (urolithiasis) and associated lower urinary tract disease
- Soft-tissue mineralization, in which calcium is deposited into the tissues
- Changes in gastrointestinal function
- Skeletal muscle contractions may be depressed, thus causing weakness
- High blood pressure (hypertension) and changes in heart-muscle contractions

EXPECTED COURSE AND PROGNOSIS

- Depends on cause of high levels of calcium in the blood (hypercalcemia) and extent of disease



KEY POINTS

- Excessive or high levels of calcium in the blood (known as “hypercalcemia”)
- Patients with underlying cancer, kidney failure, or low levels of steroids produced by the adrenal glands (known as “hypoadrenocorticism”) generally appear ill
- Signs become apparent when high levels of calcium in the blood (hypercalcemia) are serious and long-term (chronic); excessive urination (known as “polyuria” or “PU”) and excessive thirst (known as “polydipsia” or “PD”)—most common in dogs; sluggishness (lethargy)—most common in cats
- Very high levels of calcium in the blood (hypercalcemia) are a medical emergency

HYPERESTROGENISM (ESTROGEN TOXICITY)



BASICS

OVERVIEW

- “Hyperestrogenism” refers to a condition in which excessive estrogen is present in the body
- A syndrome characterized by high serum concentration of estrogens (estradiol, estriol, or estrone)
- Estrogens are hormones that are produced by the female (ovary, placenta), male (testicles), by both sexes (adrenal glands), and by some plants; most commonly recognized as a female sex hormone that is responsible for normal sexual behavior and development and function of the female reproductive tract; in the male, estrogens are responsible for Leydig cell function, which produce testosterone
- Hyperestrogenism may occur as a result of excessive estrogen secretion in the body or administration of estrogen-containing medications, such as diethylstilbestrol

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Mean Age and Range

- Older female dogs (secondary to tumors of the ovaries)
- Young female dogs (secondary to follicular ovarian cysts, in which the area containing the egg [known as the “follicle”] in the ovary develops, but ovulation does not occur normally)
- Older male dogs (secondary to tumors of the testicles)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Attractive to male dogs
- Infertility
- Prolonged heat cycle (specifically proestrus and estrus)
- Decreased libido
- Impulse to engage in sexual behavior (known as “nymphomania”)
- Bleeding from the vulva (the external genitalia of females)
- Blood in the urine (known as “hematuria”), in association with benign prostatic enlargement (known as “benign prostatic hyperplasia” or “BPH”) or low platelet count (known as “thrombocytopenia”) that may result from effect of estrogen on bone marrow; “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- Skin—non-itchy, symmetrical hair loss (known as “alopecia”); “stud dog tail”; darkened skin (known as “hyperpigmentation”)
- Reproductive abnormalities in females—fluid build-up of the vulva (the external genitalia of females); discharge from the vulva; enlargement or development of the mammary glands
- Reproductive abnormalities in males—tumor or mass in the testicles; difference in size of the testicles; decrease in size of the testicle (known as “testicular atrophy”); enlarged prostate; excessive development of the male mammary glands (known as “gynecomastia”)
- Pale gums and other moist tissues of the body (known as “mucous membranes”)
- Small, pinpoint areas of bleeding (known as “petechia”)
- Fever (due to secondary bacterial infection in association with low white-blood cell counts [known as “neutropenia”] that may result from effect of estrogen on bone marrow)
- Depression

CAUSES AND RISK FACTORS

- Follicular ovarian cysts, in which the area containing the egg (known as the “follicle”) in the ovary develops, but ovulation does not occur normally
- Tumors of the ovaries
- Tumors of the testicles (specifically Sertoli cell tumor, but also may occur secondary to Leydig and interstitial cell tumors)
- Administration of estrogen-containing medications



TREATMENT

HEALTH CARE

- Discontinue administration of estrogen-containing medications, if applicable

SURGERY

- Treatment of choice for excessive levels of estrogen (hyperestrogenism) in the intact female and male is surgical neutering; perform a “spay” or “ovariohysterectomy” for females and perform a “neuter” or “castration” for males
- Surgical removal of only one ovary containing a tumor for females or one testicle containing a tumor for males may be considered in valuable breeding animals



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Administration of antibiotics and blood products, as needed for the individual patient
- Medications (such as synthetic erythropoietin, granulocyte stimulating factor) may be considered to stimulate production of red-blood cells and/or white-blood cells in the bone marrow; lithium reportedly has been of benefit in cases of estrogen-induced lack of production of blood cells (known as “bone-marrow aplasia”)
- Administration of iron dextran intramuscularly or multiple daily doses of oral iron—necessary to support red-blood cell regeneration by the bone marrow
- Gonadotropin-releasing hormone (hormone that causes release of luteinizing hormone from the pituitary gland; “luteinizing hormone” is a female hormone that stimulates the ovarian follicle to develop and rupture to allow release of the egg and to produce progesterone)—may induce ovulation in cases of follicular ovarian cysts; however, results are unreliable



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat serial complete blood count (CBC) analyses—to evaluate response to therapy and progression of disease
- Repeat serial bone-marrow evaluations—to evaluate bone-marrow response including production of red-blood cells, white-blood cells, and platelets; signs of regeneration of blood cells may not occur for weeks to months
- Evaluation of serum progesterone (female hormone, which supports and maintains pregnancy) concentration—may be used to evaluate ovulation; serum progesterone concentration greater than 2 ng/dl (usually greater than 5 ng/dl) supports probability that ovulation has occurred
- Clinical signs of male feminization syndrome should resolve within 2 to 6 weeks after surgical removal of a tumor of the testicles

POSSIBLE COMPLICATIONS

- Decreased production of blood cells by the bone marrow (known as “bone-marrow hypoplasia”) or lack of production of blood cells (known as “bone-marrow aplasia”)
- Death

EXPECTED COURSE AND PROGNOSIS

- Lack of resolving low blood-cell counts and continued lack of response of the bone-marrow 3 weeks after surgical removal of ovarian or testicular tumors or removal of follicular ovarian cysts (in which the area containing the egg [known as the “follicle”] in the ovary develops, but ovulation does not occur normally)—associated with a grave prognosis



KEY POINTS

- Excessive levels of estrogen in the body (hyperestrogenism) may cause several conditions, many of which are serious or even potentially life-threatening
- Complications include decreased production of blood cells by the bone marrow (bone-marrow hypoplasia) and lack of production of blood cells (bone-marrow aplasia)

HYPERLIPIDEMIA

(PRESENCE OF LARGE AMOUNT OF LIPIDS [CHOLESTEROL AND TRIGLYCERIDES] IN THE BLOOD)



BASICS

OVERVIEW

- Concentration of lipids (cholesterol and triglycerides) in the blood of a fasted patient (in which food has been withheld for at least 12 hours) that exceeds the upper limit of “normal” for that species, as determined by blood tests; includes both high levels of cholesterol in the blood (known as “hypercholesterolemia”) and high levels of triglyceride in the blood (known as “hypertriglyceridemia”)
- “Lipemia”—presence of abnormally large amount of lipids in the circulating blood; serum or plasma separated from blood contains an excess concentration of triglycerides (greater than 200 mg/dL), which gives the serum or plasma a cloudy appearance
- “Lactescence”—opaque, milk-like appearance of serum or plasma that contains an even higher concentration of triglycerides (greater than 1000 mg/dL) than lipemic serum

GENETICS

- Genetic predisposition in miniature schnauzer (dog) and Himalayan (cat) for hereditary hyperlipidemias

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog and cat

Breed Predilections

- Hereditary hyperlipidemias in miniature schnauzer (dog) and Himalayan (cat)
- High levels of cholesterol in the blood for unknown reason (known as “idiopathic hypercholesterolemia”) observed in families of Doberman pinschers and rottweilers

Mean Age and Range

- Hereditary hyperlipidemias—age of onset is greater than 4 years in predisposed breeds of dog (such as the miniature schnauzer) and greater than 8 months in cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Recent ingestion of a meal
- Seizures
- Abdominal pain and distress
- Nervous system disorders (known as “neuropathies”)
- Lipemia retinalis (condition in which the blood vessels in the back of the eye [retina] appear pink rather than normal red; pink color is caused by the whitish lipids mixing with the blood)
- Lipemic aqueous (the “aqueous humor” is the transparent liquid that fills the front part of the eyeball; with high levels of triglycerides, the transparent fluid becomes cloudy—“lipemic aqueous”)
- Cutaneous xanthoma (benign nodular lesions in the skin, associated with high levels of lipids [cholesterol and triglycerides])

CAUSES

Increased Absorption of Triglycerides or Cholesterol

- Postprandial (following a meal)

Increased Production of Triglycerides or Cholesterol

- Nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin)
- Pregnancy
- Defects in lipid-clearance enzymes or lipid-carrier proteins
- High levels of chylomicrons (lipid droplets containing cholesterol esters and triglycerols) in the blood for unknown cause (condition known as “idiopathic hyperchylomicronemia”)
- High levels of chylomicrons (lipid droplets containing cholesterol esters and triglycerols) in the blood (known as “hyperchylomicronemia”) in cats
- High levels of cholesterol in the blood for unknown cause (condition known as “idiopathic hypercholesterolemia”)

Decreased Clearance or Removal of Triglycerides or Cholesterol

- Low or inadequate levels of thyroid hormone (known as “hypothyroidism”)
- Excess levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Diabetes mellitus (“sugar diabetes”)
- Inflammation of the pancreas (known as “pancreatitis”)
- Cholestasis (condition in which the flow of bile is decreased or stopped)

RISK FACTORS

- Obesity
- High dietary intake of fats
- Genetic predisposition in miniature schnauzer (dog) and Himalayan (cat)
- High levels of cholesterol in the blood for unknown cause (condition known as “idiopathic hypercholesterolemia”) observed in families of Doberman pinschers and rottweilers



TREATMENT

HEALTH CARE

- Depends on underlying cause

DIET

- Initial management is dietary
- Diet should contain less than 10% fat (for example, Hill’s Prescription Diet® r/d®, IAMS® Restricted-Calorie™ Formula)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medications may be tried, if diet fails to control the high levels of lipids (cholesterol and triglyceride) in the blood
- Gemfibrozil
- Fish oils—linolenic acid (omega-3 polyunsaturated fat)
- Clofibrate and niacin—not currently recommended in dogs or cats



FOLLOW-UP CARE

PATIENT MONITORING

- Keep triglyceride concentrations less than 500 mg/dL to avoid possibly fatal episodes of sudden (acute) inflammation of the pancreas (pancreatitis)
- Checking cholesterol often is not necessary, because high levels of cholesterol in the blood (hypercholesterolemia) are not associated with clinical signs in dogs and cats

POSSIBLE COMPLICATIONS

- Inflammation of the pancreas (pancreatitis) and seizures are common complications of high levels of lipids in the blood (hyperlipidemia) in the miniature schnauzer
- Xanthoma formation (benign nodular lesions in the skin, associated with high levels of lipids [cholesterol and triglycerides]); lipemia retinalis (condition in which the blood vessels in the back of the eye [retina] appear pink rather than normal red; pink color is caused the whitish lipids mixing with the blood); and nervous-system disorders (neuropathies) have been reported in cats with an inherited condition characterized by high levels of chylomicrons (lipid droplets containing cholesterol esters and triglycerols) in the blood (known

as “hereditary hyperchylomicronemia”); nervous-system disorders involving the limbs (known as “peripheral neuropathies”) usually resolve 2 to 3 months after institution of a low-fat diet

EXPECTED COURSE AND PROGNOSIS

- Depends on underlying cause



KEY POINTS

- Concentration of lipids (cholesterol and triglycerides) in the blood of a fasted patient (in which food has been withheld for at least 12 hours) that exceeds the upper limit of “normal” for that species, as determined by blood tests; includes both high levels of cholesterol in the blood (known as “hypercholesterolemia”) and high levels of triglyceride in the blood (known as “hypertriglyceridemia”)
- Initial management is dietary
- Diet should contain less than 10% fat (for example, Hill’s Prescription Diet® r/d®, IAMS® Restricted-Calorie™ Formula)
- Inflammation of the pancreas (pancreatitis) and seizures are common complications of high levels of lipids in the blood (hyperlipidemia) in the miniature schnauzer

HYPERPARATHYROIDISM

(EXCESSIVE LEVELS OF PARATHYROID HORMONE IN THE BLOOD)



BASICS

OVERVIEW

- “Hyperparathyroidism” is an abnormal condition in which high levels of parathyroid hormone (also known as “parathormone” or “PTH”) are circulating in the blood; “parathyroid hormone” regulates calcium and phosphorus levels in the blood—it increases calcium levels by causing calcium to be reabsorbed from bone
- “Primary hyperparathyroidism” refers to a condition in which a tumor in the parathyroid gland produces excessive levels of parathyroid hormone, leading to increased blood calcium levels
- “Secondary hyperparathyroidism” can be caused by a deficiency of calcium and vitamin D associated with malnutrition or long-term (chronic) kidney disease
- The “parathyroid glands” are small, hormone-secreting glands that are located on or near the thyroid glands; thus the name, as “para-” refers to “adjacent” or “alongside” and “thyroid” refers to the thyroid gland; the thyroid and parathyroid glands are located in the neck, near the windpipe or trachea

GENETICS

- None known for primary hyperparathyroidism, but its association with certain breeds suggests a possible hereditary basis in some cases
- Secondary hyperparathyroidism can develop in association with hereditary kidney disease (known as “hereditary nephropathy”), but is not inherited per se

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Keeshond
- Siamese

Mean Age and Range

- Dogs—mean age, 10 years; range, 5 to 15 years of age
- Cats—mean age, 13 years; range, 8 to 15 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Most dogs and cats with primary hyperparathyroidism do not appear ill
- Signs usually are mild and are due solely to the effects of high levels of calcium in the blood (known as “hypercalcemia”)
- Increased urination (known as “polyuria”)
- Increased thirst (known as “polydipsia”)
- Lack of appetite (known as “anorexia”)
- Sluggishness (lethargy)
- Vomiting
- Weakness
- Presence of stones (known as “uroliths”) in the urinary tract (condition known as “urolithiasis”)
- Stupor and coma
- Veterinarian may be able to feel enlarged parathyroid glands in the neck of cats
- Nutritional secondary hyperparathyroidism sometimes is associated with bone fractures and general poor body condition; “nutritional secondary hyperparathyroidism” is caused by diets that have too little calcium and vitamin D or too much phosphorus—it is a type of malnutrition

CAUSES

- Primary hyperparathyroidism—PTH-secreting tumor (known as an “adenoma”) of the parathyroid gland; in most cases only one gland has a tumor; malignant tumors of the parathyroid glands are uncommon
- Secondary hyperparathyroidism related to malnutrition—nutritional deficiency of calcium and vitamin D or nutritional excess of phosphorus

- Secondary hyperparathyroidism related to long-term (chronic) kidney disease—calcium is lost through the kidneys and absorption of calcium is reduced through the intestinal tract due to deficiency in a hormone (known as “calcitriol”) produced by the kidneys or phosphorus is retained in the body

RISK FACTORS

- Primary hyperparathyroidism—unknown
- Secondary hyperparathyroidism—calcium/vitamin D malnutrition or long-term (chronic) kidney disease



TREATMENT

HEALTH CARE

- Primary hyperparathyroidism generally requires inpatient care and surgery
- Secondary hyperparathyroidism related to malnutrition or long-term (chronic) kidney disease in non-critical patients can be managed on an outpatient basis

ACTIVITY

- No alterations recommended

DIET

- Calcium supplementation for secondary hyperparathyroidism, under the direction of your pet’s veterinarian
- Low phosphorus diets for secondary hyperparathyroidism related to long-term (chronic) kidney disease, as directed by your pet’s veterinarian

SURGERY

- Surgery is the treatment of choice for primary hyperparathyroidism and is often important in establishing the diagnosis
- Surgical removal of a tumor of the parathyroid gland (surgical removal of a parathyroid gland known as a “parathyroidectomy”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No medical treatment exists for primary hyperparathyroidism itself, rather treatment is directed at high levels of calcium in the blood (hypercalcemia)
- Normal saline is the fluid of choice for treatment of high levels of calcium in the blood (hypercalcemia)
- Medications to remove excess fluids from the body (known as “diuretics,” such as furosemide) and steroids can be useful in treating high levels of calcium in the blood (hypercalcemia)
- Secondary hyperparathyroidism related to long-term (chronic) kidney disease is sometimes treated with the hormone, calcitriol, but its use has not gained wide acceptance
- A new class of medications that mimic calcium to lower parathyroid hormone levels (known as “calcimimetic drugs”) is being used to treat secondary hyperparathyroidism related to long-term (chronic) kidney disease in people, but studies of these drugs in dogs and cats have not been reported
- Pamidronate is a bisphosphonate used to prevent bone loss and osteoporosis; it has been used to treat high levels of calcium in the blood (hypercalcemia) of various causes in dogs and cats
- Postoperative low levels of calcium in the blood (known as “hypocalcemia”) requires treatment with vitamin D and calcium supplements; the hormone, calcitriol, is recommended



FOLLOW-UP CARE

PATIENT MONITORING

- Postoperative low levels of calcium in the blood (hypocalcemia) is relatively common after surgical removal of one or more parathyroid glands for treatment of primary hyperparathyroidism, especially in patients with presurgical serum calcium concentrations greater than 14 mg/dl; check serum calcium concentrations once or twice daily for 1 week after surgery
- Ionized calcium levels should be monitored to guide dosage adjustments when treating postoperative low levels of calcium (hypocalcemia) with vitamin D, calcium supplements, or calcitriol
- Check blood work (serum concentrations of urea nitrogen and creatinine) in patients with kidney disease

PREVENTIONS AND AVOIDANCE

- No strategies exist for prevention of primary hyperparathyroidism
- Secondary hyperparathyroidism related to malnutrition is prevented by proper nutrition

POSSIBLE COMPLICATIONS

- Irreversible kidney failure secondary to high levels of calcium in the blood (hypercalcemia)
- Low concentration of calcium in the blood (hypocalcemia) is a potential complication of surgical removal of the parathyroid gland (parathyroidectomy)

EXPECTED COURSE AND PROGNOSIS

- Untreated disease usually progresses to end-stage kidney or nervous system disease
- Prognosis for surgical treatment of tumors of the parathyroid gland (parathyroid adenoma) is excellent
- Recurrence is seen in a small percentage of cases
- In animals that develop postoperative low levels of parathyroid hormone (known as “hypoparathyroidism”), the return of normal parathyroid gland function is unpredictable and can take weeks to months



KEY POINTS

- Signs generally are related to changes in calcium status of the body
- Low concentration of calcium in the blood (hypocalcemia) is a potential complication of surgical removal of the parathyroid gland (parathyroidectomy)

PULMONARY HYPERTENSION

(HIGH BLOOD PRESSURE IN THE LUNGS)



BASICS

OVERVIEW

- Elevation in systolic blood pressure in the arteries of the lungs (defined as pulmonary artery pressure greater than 30 mmHg or mean pulmonary arterial pressure greater than 20 mmHg)
- Several events can lead to elevations in blood pressure in the arteries of the lungs (pulmonary artery pressure)—primary pulmonary hypertension, a congenital (present at birth) abnormality in the blood vessels of the lungs has not been identified in dogs or cats; secondary pulmonary hypertension can be caused by narrowing or constriction of the arteries or capillaries of the lungs, blockage of the artery of the lung (pulmonary artery), high pressure within the left atrium (left-sided top chamber of the heart) with resultant increase in blood pressure in the capillaries of the lungs, or excessive blood flow in the arteries of the lungs
- Many of the causes for development of high blood pressure in the arteries of the lungs (pulmonary hypertension) involve the heart; the heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)

GENETICS

- No genetic basis found
- High blood pressure in the arteries of the lungs (pulmonary hypertension) can be secondary to several congenital (present at birth) heart defects that may have a genetic basis

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- May be based on underlying cause of high blood pressure in the arteries of the lungs (pulmonary hypertension), such as congenital (present at birth) heart disease

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs may be due to high blood pressure in the arteries of the lungs (pulmonary hypertension) or to the underlying primary disease
- Exercise intolerance
- Difficulty breathing (known as “dyspnea”)
- Coughing; spitting up of blood derived from the lungs due to pulmonary or bronchial hemorrhage (known as “hemoptysis”)
- Fainting (known as “syncope”)
- Abdominal distention
- Weight loss
- Heart murmur
- Abnormal heart and/or lung sounds heard when listening to the chest with a stethoscope
- Bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”)
- Distention of the jugular veins in the neck

CAUSES

Lung Disease

- Blockage of blood flow in the lungs
- Pneumonia
- Long-term (chronic) inflammation of the bronchi (airways going from the windpipe [trachea] into the lungs; condition known as “bronchitis”)
- Inflammation of the bronchi characterized by the presence of eosinophils, a type of white-blood cell involved in allergic responses by the body and active in fighting larvae of parasites (condition known as “eosinophilic bronchitis”)
- Widespread tumors in the lungs

- Adult respiratory distress syndrome, a group of lung abnormalities that develop secondary to various serious illnesses that cause sudden breathing difficulties

Blood Clots in the Lungs (known as “pulmonary thromboembolism”)

- Excessive levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Condition in which proteins are lost from the body through the kidneys (known as “protein-losing nephropathy”)
- Presence of pus-forming bacteria and their poisons in the blood or tissues (condition known as “sepsis”)
- Heartworm disease
- Immune-mediated hemolytic anemia—accelerated destruction or removal of red-blood cells related to an immune response, in which the body produces antibodies against red-blood cells
- Cancer
- Inflammation of the pancreas (known as “pancreatitis”)
- Inflammation/infection of the lining of the heart (known as “endocarditis”)
- Blood-clotting disorder (known as “disseminated intravascular coagulation” or “DIC”)
- Primary heart disease

Heartworm Disease

Congenital (Present at Birth) Heart Disease

- Various heart defects (such as patent ductus arteriosus, ventricular septal defect, atrial septal defect)
- Probably a rare cause of high blood pressure in the arteries of the lungs (pulmonary hypertension)

Left-Sided Heart Disease

- Backward flow of blood through the mitral valve (known as “mitral regurgitation”)
- Heart muscle is flabby and weak (known as “dilated cardiomyopathy”)
- Heart muscle disease characterized by inappropriate enlargement or thickening of the heart muscle of the left ventricle (known as “hypertrophic cardiomyopathy”)
- Heart muscle disease in which the muscle is “stiff” and does not expand, such that blood cannot fill the ventricles normally (known as “restrictive cardiomyopathy”)
- Narrowing of the mitral valve (known as “mitral stenosis”)
- Tumors of the left atrium

Causes of Long-Term (Chronic) Low Levels of Oxygen in the Body (known as “hypoxia”)

- Decreased movement of air into and out of the lungs (known as “hypoventilation”)
- High-altitude disease

RISK FACTORS

- Heart and lung disease
- Heartworm disease
- Diseases associated with blood clots to the lungs (pulmonary thromboembolism)
- Obesity
- High altitude



TREATMENT

HEALTH CARE

- Hospitalize patients with severe breathing distress, until stable
- Administer oxygen therapy, medications to enlarge or dilate the bronchi (known as “bronchodilators”), medications to remove excess fluid from the body (known as “diuretics”), and antibiotics on an emergency basis in accordance with underlying disease
- Monitor hydration and body temperature closely
- Administer fluid therapy carefully on basis of hydration status and severity of right-sided heart disease; right-sided heart disease may contraindicate fluid therapy
- Maintain low-stress environment
- Medical management is controversial; treatment of the primary underlying disease process should be the focus, if possible

ACTIVITY

- Restricted

DIET

- Specific guidelines based on underlying disease
- If in heart failure, restricted sodium diet may have benefit

SURGERY

- Surgical heartworm extraction is a consideration in patients with severe infestation



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- The ideal therapeutic agent should reduce blood pressure in the lungs without affecting the general (systemic) circulation; oxygen can accomplish this, but long-term oxygen administration is not feasible in these patients; short-term or intermittent use of oxygen may be beneficial

Medications to Enlarge or Dilate Blood Vessels (Known as “Vasodilators”)

- Ideally, base selection on lung and systemic blood pressure responses during heart catheterization
- Choices include angiotensin-converting enzyme (ACE) inhibitors (such as enalapril, benazepril), hydralazine, and calcium-channel blockers
- Often not useful due to development of generalized (systemic) low blood pressure (known as “hypotension”)
- Sildenafil (Viagra®) is known to cause enlargement of lung blood vessels with minimal enlargement of generalized (systemic) blood vessels; the usefulness of this medication in dogs is under investigation

Medications to Enlarge or Dilate Bronchi (Bronchodilators)

- May benefit treatment of low oxygen (hypoxia)-mediated high blood pressure in the arteries of the lungs (pulmonary hypertension)
- Choices include sympathomimetics (such as terbutaline) and methylxanthines (such as theophylline, aminophylline)
- Bronchodilators may have additional positive effects on contraction of heart muscle (known as “positive inotropes”)

Medications to Increase Contraction of Heart Muscle (Positive Inotropes)—Digoxin, Dobutamine

- Not a primary treatment of high blood pressure in the arteries of the lungs (pulmonary hypertension)
- May improve right heart function and resolve congestive heart failure
- Monitor closely for digoxin-related irregular heart beats (known as “arrhythmias”)

Medications to Prevent Blood Clots (Known as “Anticoagulant Therapy”)

- Indicated if blood clots in the lungs (pulmonary thromboembolism) are diagnosed
- Choices include heparin, warfarin, and aspirin
- Low molecular-weight heparin (enoxaparin) currently is under investigation in animals



FOLLOW-UP CARE

PATIENT MONITORING

- Physical examination with careful listening of the heart and lungs with a stethoscope (known as “auscultation”)
- Monitor for worsening of clinical signs
- Chest X-rays
- Arterial blood gases (measurements of oxygen and carbon dioxide levels in arterial blood)
- Echocardiogram (use of ultrasound to evaluate the heart and major blood vessels)
- Electrocardiogram (“ECG,” a recording of the electrical activity of the heart)

PREVENTIONS AND AVOIDANCE

- Early evaluation and prevention of conditions that increase likelihood of development of high blood pressure in the arteries of the lungs (pulmonary hypertension)

POSSIBLE COMPLICATIONS

- Right-sided heart failure
- Fainting (syncope)
- Irregular heart beats (arrhythmias)
- Sudden death

EXPECTED COURSE AND PROGNOSIS

- Based on ability to reverse underlying disease
- When changes are irreversible, treatment is designed to improve patient's condition rather than to cure the disease
- In general, prognosis is very guarded



KEY POINTS

- Diagnosis often is presumptive (that is, based on clinical signs); for definitive diagnosis, need heart catheterization and/or Doppler echocardiography (use of ultrasound to evaluate the heart and major blood vessels)
- Prognosis varies with reversibility of the underlying disease, but is very guarded in most cases
- Avoid environments that may increase likelihood of breathing distress—excessively cold or dry air, excessive heat, second-hand smoke, high altitudes

SYSTEMIC HYPERTENSION

(HIGH BLOOD PRESSURE THROUGHOUT THE BODY)



BASICS

OVERVIEW

- “Systemic” refers to the entire body versus individual parts of the body; “hypertension” is high blood pressure
- “Systemic hypertension” is a sustained elevation in systolic or diastolic (or both) arterial blood pressure; if no other disease process is identified to cause hypertension, it is called “primary hypertension,” “essential hypertension” or “idiopathic hypertension”—“idiopathic” refers to “unknown cause;” if the hypertension is due to another disease process, it is termed “secondary hypertension”
- Secondary hypertension is more common in veterinary medicine; cause of primary hypertension is not fully understood, but some cases have a hereditary component
- Elevations in blood pressure may be related to a measurement artifact (stress-induced or “white-coat” effect [that is, being in the animal hospital and being nervous around the veterinary staff]) or disease
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)

GENETICS

- Colonies of dogs with high blood pressure have been produced by mating dogs with primary (essential or idiopathic) hypertension; mode of inheritance is not known

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Dogs—mean age, 8.9 years; range, 2 to 14 years of age
- Cats—mean age 15.1 years; range, 4 to 20 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden (acute) blindness
- Bleeding within the eye
- Dilated pupils
- Separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid;” condition known as “retinal detachment”); the “retina” contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision
- Swollen or shrunken kidneys
- Blood in the urine (known as “hematuria”); presence of protein in the urine (known as “proteinuria”)—protein in the urine is detected by diagnostic tests (such as dipstick, microalbuminuria testing, and urine protein-to-creatinine ratio) performed as part of the urine evaluation
- Bleeding in the nose and nasal passages (known as “epistaxis” or a “nosebleed”)
- Seizures; disorientation; wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”); circling; weakness or partial paralysis; short, rapid movements of the eyeball (known as “nystagmus”)
- Heart murmurs and sequence of three heart sounds (known as a “gallop rhythm”), when listening to the heart with a stethoscope; gallop rhythm is heart beats that sound like a galloping horse instead of normal “lub-dub”
- Pet may be in congestive heart failure; signs include cough; difficulty breathing (known as “dyspnea”); bluish discoloration of the skin and moist tissues of the body caused by inadequate oxygen levels in the red-blood cells (discoloration known as “cyanosis”); “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Enlarged thyroid gland may be present in cats, when increased levels of thyroid hormone (known as “hyperthyroidism”) is cause of high blood pressure (hypertension)

CAUSES

Primary or Essential Hypertension

- Not known

Secondary Hypertension

- Accounts for over 80% of cases
- Kidney disease
- Increased levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Increased levels of thyroid hormones (hyperthyroidism)
- Diabetes mellitus (“sugar diabetes”)—uncommon cause of high blood pressure (hypertension)
- Pheochromocytoma—rare tumor of the adrenal gland
- Increased levels of aldosterone, the hormone that regulates sodium and potassium in the body (condition known as “hyperaldosteronism”)—rare condition, usually due to a tumor in the adrenal gland
- Central nervous system disease—rare cause



TREATMENT

HEALTH CARE

- If possible, treat as an outpatient
- Hospitalization may be stressful to the patient
- Inpatient care may be necessary depending upon the underlying condition (such as the need for fluid therapy in a pet with kidney failure) or serious complications related to high blood pressure (hypertension), such as nervous system signs

ACTIVITY

- Depends on condition and underlying cause

DIET

- Influenced by underlying cause; salt (sodium) restriction considered controversial and, if used alone, is unlikely to lower blood pressure
- Avoid high salt intake

SURGERY

- Dictated by underlying cause; may be indicated for some underlying causes



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treat underlying cause
- Dogs—medications to decrease blood pressure, such as angiotensin-converting enzyme (ACE) inhibitor (example, enalapril or benazepril) or calcium channel blocker (example, amlodipine or diltiazem)
- Cats—medications to decrease blood pressure, such as calcium channel blocker (example, amlodipine) or angiotensin-converting enzyme (ACE) inhibitor (example, benazepril)
- β -blocker (example, propranolol or atenolol) or α -adrenergic blocker, if no response to ACE inhibitor or calcium channel blocker
- In emergency management, hydralazine or enalaprilat can be used in dogs; continuous direct blood-pressure monitoring is recommended
- Medications to enlarge or dilate blood vessels, such as hydralazine (dogs and cats) or phenoxybenzamine (cats)



FOLLOW-UP CARE

PATIENT MONITORING

- Blood pressure measurements of less than 150/95 may be considered the goal blood pressure
- Blood pressure and complications of high blood pressure (especially changes in the back of the eye [retina; changes known as “retinopathy”]) checked weekly until blood pressure is controlled
- Laboratory tests to measure side effects of medications and clinical disease response

POSSIBLE COMPLICATIONS

- Congestive heart failure, a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Kidney disease
- Kidney failure
- Degeneration of the retina (back part of the eye) due to high blood pressure (known as “hypertensive retinopathy”)
- Bleeding in the retina or separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)
- Bleeding or blockage of blood flow in the brain (known as a “cerebral vascular accident”) leading to various central nervous system signs

EXPECTED COURSE AND PROGNOSIS

- Dictated by underlying cause
- Blood pressure can be controlled with appropriate therapy in most patients, but therapy does not necessarily improve survival time



KEY POINTS

- Unless underlying cause is curable or controllable, patient is likely to be on medications to control blood pressure (known as “antihypertensive medication”) indefinitely
- Uncontrolled high blood pressure (hypertension) can lead to various medical conditions (such as bleeding in the back of the eye [retina]; separation of the back part of the eye from the underlying, vascular part of the eye ball (retinal detachment); progressive kidney damage; heart disease; or nervous system signs)

INCREASED BODY TEMPERATURE (HYPERTHERMIA) AND HEAT STROKE



BASICS

OVERVIEW

- “Hyperthermia” is an elevation in body temperature above the generally accepted normal range of body temperatures; although published normal values for dogs and cats vary slightly, it usually is accepted that body temperatures above 103° F (39° C) are abnormal
- Hyperthermia can be categorized into “fever” and “non-fever” hyperthermia; “fever” hyperthermia results from inflammation in the body (such as secondary to a bacterial infection); “non-fever” hyperthermia results from all other causes of increased body temperature
- “Heat stroke” is a form of “non-fever” hyperthermia that occurs when heat-dissipating mechanisms of the body cannot accommodate excessive heat; heat stroke can lead to multiple organ dysfunction
- Temperatures of 106° F (41° C) or higher, without signs of inflammation are suggestive of “non-fever” hyperthermia
- “Malignant hyperthermia” is an uncommon familial (runs in certain families or lines of animals) “non-fever” hyperthermia that can occur secondary to some anesthetic agents
- Other causes of “non-fever” hyperthermia include excessive exercise, thyrotoxicosis (excessive levels of thyroid hormones in the body), and lesions in the hypothalamus, the part of the brain that regulates body temperature

The following information primarily relates to “non-fever” hyperthermia:

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and uncommonly cats

Breed Predilection

- May occur in any breed
- Long-haired animals
- Short-nosed, flat-faced (known as “brachycephalic”) breeds

Mean Age and Range

- All ages, but often age extremes
- Young dogs may tend to overexert
- Old dogs with preexisting disease

SIGNS/OBSERVED CHANGES in the ANIMAL

- Identifiable underlying cause, such as a hot day, being locked in car or other confined area without adequate ventilation, grooming accident associated with drying cages, excessive exercise, restricted access to water
- Underlying disease that increases likelihood of developing hyperthermia, such as paralysis of the voice box or larynx (known as “laryngeal paralysis”), heart and/or blood vessel disease, nervous system and/or muscular disease, previous history of heat-related disease
- Panting
- Excessive drooling (known as “hypersalivation”)
- Increased body temperature (hyperthermia)
- Reddened gums and moist tissues of the body (known as “hyperemic mucous membranes”)
- Rapid heart rate (known as “tachycardia”)
- Irregular heart beats (known as “arrhythmias”)
- Shock
- Breathing distress
- Vomiting blood (known as “hematemesis”)
- Passage of blood in the bowel movement or stool (known as “hematochezia”)
- Black, tarry stools (due to the presence of digested blood; condition known as “melena”)
- Small, pinpoint areas of bleeding (known as “petechiae”)
- Changes in mental status
- Seizures

- Muscle tremors
- Wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)
- Unconsciousness in which animal cannot be stimulated to be awakened (known as “coma”)
- Production of only small amounts of urine (known as “oliguria”) or no urine (known as “anuria”)
- Stoppage of breathing (known as “respiratory arrest”)
- Stoppage of the heart and breathing (known as “cardiopulmonary arrest”)

CAUSES

- Excessive environmental heat and humidity (may be due to weather conditions, or accidents such as being enclosed in unventilated room, car, or grooming dryer cages)
- Upper airway disease; the “upper airway” (also known as the “upper respiratory tract”) includes the nose, nasal passages, throat (pharynx), and windpipe (trachea)
- Exercise
- Poisoning; some poisonous compounds (such as strychnine and metaldehyde [slug and snail bait]) lead to seizures, which can cause increased body temperature
- Anesthesia (malignant hyperthermia)

RISK FACTORS

- Previous history of heat-related disease
- Age extremes
- Heat intolerance due to poor acclimatization
- Obesity
- Poor heart/lung conditioning
- Underlying heart/lung disease
- Increased levels of thyroid hormone (known as “hyperthyroidism”)
- Short-nosed, flat-faced (brachycephalic) breeds
- Thick hair coat
- Dehydration



TREATMENT

HEALTH CARE

- Early recognition is key
- Immediately correct increased body temperature (hyperthermia)
- Patients should be hospitalized until temperature is stabilized
- Most patients need intensive care for several days
- Treat complications, such as the blood-clotting disorder (“disseminated intravascular coagulopathy” or “DIC”), kidney failure, fluid build-up in the brain (known as “cerebral edema”)
- Treat underlying disease or correct factors that increase likelihood of developing increased body temperature, if possible

External Cooling Techniques

- Spray with water or immerse in water prior to transport to veterinary facility, as directed by your pet’s veterinarian
- Convection cooling with fans
- Evaporative cooling (such as isopropyl alcohol on foot pads, groin, and under the forelegs)
- Stop cooling procedures when temperature reaches 103° F to avoid dropping to too low a body temperature (known as “hypothermia”)
- Avoid ice, as this may cause blood vessels near the surface of the body to constrict (known as “peripheral vasoconstriction”) and may decrease heat dissipation; shivering response also is undesirable, as it creates heat

Other Care

- Continuous temperature monitoring
- Fluid therapy
- Provide oxygen supplementation via mask, cage, or nasal catheter
- Breathing or ventilatory support, if required

ACTIVITY

- Restricted

DIET

- No food or water by mouth, until the pet is stable

SURGERY

- Surgical opening into the windpipe or trachea (known as a “tracheostomy”) may be required, if upper airway obstruction is an underlying cause or a contributing factor



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No specific drugs are required for treating increased body temperature (hyperthermia) or heat stroke; therapy is dependent on clinical presentation
- Broad-spectrum antibiotics may decrease the incidence of bacteria moving from the intestinal tract into the body
- Sudden (acute) kidney failure—medications to dilate the blood vessels in the kidneys and to increase blood pressure, such as dopamine; medications to stimulate urine production (known as “diuretics”), such as furosemide
- Fluid build-up in the brain (cerebral edema)—medications to remove excess fluid from the body, such as mannitol; furosemide 30 minutes following mannitol administration; medications to decrease brain swelling and inflammation, such as steroids (examples are dexamethasone sodium phosphate, prednisone sodium succinate, and methyl prednisolone)
- Irregular heart beats (arrhythmia)—heart medications, such as lidocaine or procainamide
- Metabolic acidosis (a condition in which levels of acid are increased in the blood)—sodium bicarbonate
- Blood-clotting disorder (disseminated intravascular coagulopathy or DIC)—fresh frozen plasma and heparin
- Decreased number of platelets (known as “thrombocytopenia”)—severe thrombocytopenia can be treated with frozen platelet concentrates; “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- Vomiting or diarrhea with presence of blood—broad-spectrum antibiotics, as well as a histamine-2 (H-2) blocker (such as famotidine) in combination with sucralfate (medication that forms a protective barrier over ulcers in the gastrointestinal tract)
- Seizures—medications to control seizures, such as diazepam or phenobarbital



FOLLOW-UP CARE

PATIENT MONITORING

- Patients should be monitored closely during cool-down period and for a minimum of 24 hours post episode of hyperthermia; most animals must be monitored for several days, depending on clinical presentation and complications
- A thorough physical examination should be performed daily
- Body temperature
- Body weight
- Blood pressure
- Blood-clotting status (known as “coagulation status”)—blood tests, such as activated-clotting time (ACT), prothrombin time (PT), partial thromboplastin time (PTT), and fibrogen-degradation products (FDP)
- Electrocardiogram (“ECG,” a recording of the electrical activity of the heart)
- Listening to the chest (heart and lungs) with a stethoscope (known as “thoracic auscultation”)
- Urine output
- Urinalysis
- Packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood) and total protein (a quick laboratory test that provides general information on the level of protein in the fluid portion of the blood)
- Complete blood count (CBC) and serum biochemical profile

PREVENTIONS AND AVOIDANCE

- Avoid risk factors

POSSIBLE COMPLICATIONS

- Irregular heart beats (arrhythmias)
- Organ failure
- Unconsciousness in which animal cannot be stimulated to be awakened (coma)
- Seizures
- Sudden (acute) kidney failure
- Blood-clotting disorder (disseminated intravascular coagulation or DIC)
- Generalized (systemic) inflammatory response syndrome
- Fluid build-up in the lungs (known as “pulmonary edema”); sudden (acute) breathing distress
- Disease characterized by the breakdown of red-muscle tissue (known as “rhabdomyolysis”)
- Death of liver cells (known as “hepatocellular necrosis”)
- Stoppage of breathing (respiratory arrest)
- Stoppage of the heart and breathing (cardiopulmonary arrest)
- Death

EXPECTED COURSE AND PROGNOSIS

- Prognosis is dependent on underlying cause or disease process
- Prognosis is guarded, depending on complications that occur and duration of episode
- One episode of hyperthermia or heat stroke increases the likelihood that the pet may have other episodes because of damage to the body-temperature regulatory center of the brain



KEY POINTS

- Be aware of clinical signs, so you may respond quickly to an episode of hyperthermia or heat stroke
- Know how to cool off your pet; talk to your pet’s veterinarian for information on the appropriate procedure
- An episode of heat stroke may increase the likelihood of additional episodes

HYPERTHYROIDISM

(EXCESSIVE PRODUCTION OF THYROID HORMONE)



BASICS

OVERVIEW

- Disease condition caused by high levels of thyroid hormones that increase metabolism in the body
- The thyroid gland normally produces thyroid hormones in response to stimulation by the pituitary gland, the “master gland” of the body; the thyroid hormones normally increase chemical processes occurring within cells of the body; however, in hyperthyroidism, the excessive hormone levels put the cells and body into “overdrive”
- Thyroid hormones are known as “triiodothyronine” or “T₃” and “tetraiodothyronine” or “T₄”

GENETICS

- No known genetic predisposition

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats; most common hormonal (endocrine) disease of cats; one of the most common diseases seen in late middle-aged and old cats
- Rare in dogs

Breed Predilections

- None

Mean Age and Range

- Mean age in cats, approximately 13 years; range 4 to 22 years

SIGNS/OBSERVED CHANGES in the ANIMAL

- Involves many organ systems due to the overall increase in metabolism
- Weight loss
- Unkempt appearance
- Poor body condition
- Increased appetite (known as “polyphagia”)
- Vomiting
- Diarrhea
- Increased thirst (known as “polydipsia”)
- Rapid breathing (known as “tachypnea”)
- Difficulty breathing (known as “dyspnea”)
- Heart murmur; rapid heart rate (known as “tachycardia”); particular abnormal heart beat, known as a “gallop rhythm”
- Hyperactivity
- Aggression
- Large thyroid gland
- Thickened nails
- Less than 10% of patients are referred to as “apathetic;” these patients exhibit atypical signs (such as poor appetite, loss of appetite, depression, and weakness)

CAUSES

- Cats—autonomously hyperfunctioning thyroid nodules (where the thyroid nodules produce excess thyroid hormones outside of the control of the pituitary gland, so called “autonomous” production); rarely, thyroid cancer (known as “thyroid carcinoma”)
- Dogs—thyroid hormone (T₄ or T₃) secretion by a thyroid cancer (thyroid carcinoma)

RISK FACTORS

- Some reports have linked hyperthyroidism in cats to some canned food diets
- Advancing age increases risk



TREATMENT

HEALTH CARE

- Outpatient management usually suffices for cats, if drugs that inhibit the production of thyroid hormones (known as “antithyroid drugs”) are used
- Treatment using a radioactive form of iodine (known as “radioiodine treatment”) or surgical removal of the thyroid gland (known as “thyroidectomy”) require inpatient treatment and monitoring
- Rare cases of congestive heart failure require emergency, inpatient intensive care

ACTIVITY

- No alterations recommended

DIET

- Poor absorption of many nutrients and high metabolism suggest the need for a highly digestible diet, with high availability of protein in untreated hyperthyroidism
- Resolution of signs resulting from excessive levels of thyroid hormones in the body (condition known as “thyrotoxicosis”) eliminates the need for dietary modifications in many animals
- Dietary modification may be necessary to treat or control complications (such as kidney damage)

SURGERY

- Surgical removal of the thyroid gland (thyroidectomy) is one recommended treatment for hyperthyroidism in cats
- Surgical treatment of thyroid carcinoma (dogs and cats) is usually not curative, but can control signs (known as “palliative treatment”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Radioiodine (radioactive form of iodine) is a safe and effective treatment; use of radioactive iodine requires special facilities; availability of veterinary facilities offering this treatment is limited, but increasing
- Methimazole (Tapazole®) inhibits the production of thyroid hormones (antithyroid drug) and is recommended most often
- Methimazole can be administered through the skin (transdermally); transdermal methimazole must be prepared by a pharmacist; resolution of signs resulting from the excessive levels of thyroid hormones in the body (thyrotoxicosis) takes longer with transdermal methimazole than with methimazole given by mouth
- β -blockers—sometimes used to treat some of the heart and nervous system effects of excess thyroid hormones; can be used in combination with methimazole; mainly used to prepare the patient for surgical removal of the thyroid gland (thyroidectomy) or radioiodine therapy
- Carbimazole—another useful antithyroid drug that inhibits production of thyroid hormones; not available in the United States
- Propylthiouracil—is an antithyroid drug that inhibits production of thyroid hormones; it can be useful if methimazole is unavailable; side effects may be more common and more severe than with methimazole
- Iopodate—a radiographic contrast agent; can be used to treat some cases of mild hyperthyroidism, but not effective in most hyperthyroid patients; long-term effectiveness has not been established



FOLLOW-UP CARE

PATIENT MONITORING

- Methimazole—physical examination, complete blood count with platelet count, serum biochemical analysis, and serum thyroid hormone (T_4) determination every 2 to 3 weeks for the initial 3 months of treatment; the dosage is adjusted to maintain serum thyroid

hormone (T_4) concentration in the low-normal range

- Surgical removal of the thyroid gland (thyroidectomy)—watch for development of low blood-calcium levels (known as “hypocalcemia”) and/or paralysis of the voice box (larynx) during the initial postoperative period; measure serum thyroid hormone (T_4) concentrations in the first week of surgery and every 3 to 6 months thereafter, to check for recurrence
- Radioiodine (radioactive form of iodine)—measure serum thyroid hormone (T_4) concentrations 2 weeks after treatment and every 3 to 6 months subsequently
- Kidney function—kidney filtration rates decline following treatment in most patients; therefore, perform a physical examination, serum biochemistry, and urinalysis 1 month after treatment and then as indicated by clinical history

POSSIBLE COMPLICATIONS

- Untreated disease can lead to congestive heart failure; diarrhea that is difficult to treat; kidney damage; detachment of the retina (a layer in the back of the eye) as a result of high blood pressure (hypertension); and death
- Complications of surgical treatment include low levels of parathyroid hormone (known as “hypoparathyroidism;” the parathyroid glands are small glands adjacent to the thyroid gland, which may be removed at the time of the surgical removal of the thyroid gland); low levels of thyroid hormone (known as “hypothyroidism”); and paralysis of the voice box (larynx)
- Low levels of thyroid hormone (hypothyroidism) is rare following radioiodine therapy (radioactive form of iodine)

EXPECTED COURSE AND PROGNOSIS

- Uncomplicated disease—prognosis is excellent; recurrence is possible and most commonly is associated with poor owner compliance with medical management; regrowth of overactive thyroid tissue is possible, but uncommon after surgical removal of the thyroid gland (thyroidectomy) or radioiodine treatment (radioactive form of iodine)
- Reported mean survival time for cats treated with radioiodine is 4 years; mean survival time for cats treated with methimazole is 2 years; mean survival time for cats treated with radioiodine and methimazole is 5.3 years
- Cats with pre-existing kidney disease have a poorer prognosis; kidney failure is the most common cause of death in hyperthyroid cats
- Dogs or cats with thyroid cancer (thyroid carcinoma)—prognosis is poor; treatment with radioiodine (radioactive form of iodine), surgery, or both usually is followed by recurrence of disease; chemotherapy is of questionable benefit



KEY POINTS

- Disease condition caused by high levels of thyroid hormones that increase metabolism in the body
- Most common hormonal (endocrine) disease of cats; one of the most common diseases seen in late middle-aged and old cats
- Rare in dogs
- Potential side effects of drugs that inhibit the production of thyroid hormones (antithyroid drugs) and surgical complications
- Be aware of possible (rare) recurrence after treatment

HYPERTROPHIC OSTEODYSTROPHY (HOD)

(A BONE DISEASE OF RAPIDLY GROWING PUPPIES)



BASICS

OVERVIEW

- Disease characterized by inflammation of the metaphyseal area of bone that affects rapidly growing puppies, especially large-breed puppies
- Long bones (such as the humerus, radius and ulna in the foreleg and the femur and tibia in the rear leg) have three sections: the end of the bone, known as the “epiphysis;” the shaft or long portion of the bone, known as the “diaphysis;” and the area that connects the end and the shaft of the bone, known as the “metaphysis”
- The metaphysis is the area where bone growth occurs in puppies; the long bones in the body grow in length at specific areas known as “growth plates;” these areas usually continue to produce bone until the bones are fully developed, at which time, no further growth is needed; the growth plates then “close” and become part of the hard bone
- Disease also known as “HOD”

GENETICS

- Suspect genetic basis of over reaction to immune stimulation (such as vaccination)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Large, rapidly growing breeds
- Great Dane; Weimaraner—most common
- Reported—Irish wolfhound; St. Bernard; Kuvasz; Irish setter; Doberman pinscher; German shepherd dog; Labrador retriever; others

Mean Age and Range

- Affects puppies 3 to 4 months of age
- Range of onset of signs—2 to 8 months of age

Predominant Sex

- Males more than females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Lameness— symmetrical, more severe in forelimbs; may be episodic; degree varies from mild to non–weightbearing; initial episode may resolve without relapse
- Depend on severity of the episode
- Often a depressed puppy that is reluctant to move
- Lack of appetite—common
- Painful
- Growth areas of the long bones (metaphyses)—painful; warm; swollen
- Fever—as high as 41.1° C (106° F)
- Weight loss; may be severe with muscle wasting (known as “cachexia”)
- Dehydration
- Diarrhea
- Debilitation
- Generalized illness—respiratory or gastrointestinal

CAUSES

- Unknown; several theories have been considered—some have been eliminated as possible causes through research, while others may be involved with the disease, but have not been proven to cause the disease

The following theories have been considered:

Metabolic

- Inadequate levels of vitamin C (known as “hypovitaminosis C”)—this has been eliminated as a possible cause; disease may be a result of overuse of available Vitamin C in hyperactive bone formation
- Low levels of copper (known as “hypocuprosis”)—has been identified as a cause in rats, but not in dogs

Nutritional

- Providing too much food or food that has excessive levels of certain nutrients (known as “overnutrition”) and/or giving too many supplements (known as “oversupplementation”)—overnutrition and oversupplementation appear to be present in some affected puppies, but not all; therefore, it may play a role in some cases
- Incomplete occurrence in litters (that is, not all puppies in a litter may be affected)
- Correcting diet does not always alter the course of the disease or eliminate relapses

Infectious

- Bacterial or fungal organisms—infection may be secondary to bone involvement and not cause of disease
- An association with the timing of vaccinations has been suggested

RISK FACTORS

- Vaccination may lead to uncontrolled inflammation in the bone-forming centers (known as the “osteogenic centers”)



TREATMENT

HEALTH CARE

- None specific
- Supportive care—depends on severity of disease; care may range from none needed to intensive care, for severely affected puppies
- Depends on the severity of the episode, fever, and the puppy’s ability to maintain normal hydration and willingness to eat
- Some puppies will not stand or move—prone to develop pressure or “bed” sores; turn every 2 to 4 hours to prevent sores and to improve breathing
- Intravenous fluid therapy—for dehydration and then maintenance fluid needs

ACTIVITY

- Restricted—running and jumping may increase injury to the growth areas of the long bones (metaphyses) and result in further inflammation
- Confine to a small, well-padded area
- Leash walking only (if the puppy is able to stand and walk)

DIET

- Normal, commercial puppy ration, as directed by your pet’s veterinarian
- Avoid supplements

SURGERY

- None specific
- May need feeding tube to be placed surgically—in debilitated puppies that will not eat or drink and have frequently relapsing episodes of sudden (acute) clinical signs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs)—to control pain and fever; may try aspirin, carprofen, or etodolac, as directed by your pet’s veterinarian
- Prednisone—only when no response is seen to NSAIDs
- Vitamin C—may be inadvisable as it may make condition worse; may speed up abnormal calcification of affected bone and may

decrease bone remodeling



FOLLOW-UP CARE

PATIENT MONITORING

- Signs of improvement—less sensitivity to the growth areas of the long bones (metaphyses); patient gets up; appetite improves; fever resolves

POSSIBLE COMPLICATIONS

- Severe weight loss with muscle wasting (cachexia)
- Permanent bowing deformities of the limbs
- Secondary bacterial infection
- Pressure or “bed” sores
- Involuntary muscle twitching, seizures—with low levels of calcium in the blood (known as “hypocalcemia”)
- May see secondary generalized disease caused by the spread of bacteria in the blood (known as “septicemia”)

EXPECTED COURSE AND PROGNOSIS

- Course—days to weeks
- Most patients—one or two episodes and recover
- Some patients—have relapsing episodes of pain and fever that do not respond to treatment; rarely die or are euthanized
- Prognosis—usually good; guarded with multiple relapses or complicating secondary problems
- Persistent bowing deformity of the limbs—eliminates many purebred puppies from the show ring



KEY POINTS

- Disease characterized by inflammation of the metaphyseal area of the bone that affects rapidly growing puppies, especially large-breed puppies
- Lameness—symmetrical, more severe in forelimbs; may be episodic; degree varies from mild to non-weightbearing
- Disease tends to relapse
- Bony deformities will remodel to some degree with time, but bowing of the limbs and twisting or bending of the bones outward, away from the center of the body (known as “valgus angular deformity”) are permanent
- The more severe the disease, the more severe the bowing deformity

HYPOADRENOCORTICISM OR ADDISON'S DISEASE

(INADEQUATE PRODUCTION OF HORMONES BY THE ADRENAL GLANDS)



BASICS

OVERVIEW

- A hormonal disorder resulting from decreased production of hormones (glucocorticoids and/or mineralocorticoids) by the adrenal glands
- “Glucocorticoids” are a class of hormones produced by the adrenal glands; they typically are called “steroids;” glucocorticoids are involved in metabolism and the stress response and they have anti-inflammatory properties
- “Mineralocorticoids” are another class of hormones produced by the adrenal glands; they are involved in regulation of salt (sodium and potassium) in the body; “aldosterone” is a mineralocorticoid that regulates sodium and potassium in the body
- Addison’s disease refers to decreased production of both glucocorticoids and mineralocorticoids
- Glucocorticoid (cortisol) deficiency contributes to lack of appetite (known as “anorexia”); vomiting; black, tarry stools (due to the presence of digested blood; condition known as “melena”); sluggishness (lethargy); and weight loss
- Inadequate glucocorticoid levels increase likelihood of the patient developing low blood glucose or sugar (known as “hypoglycemia”)
- Mineralocorticoid (aldosterone) deficiency results in an inability to retain sodium in the body and to excrete potassium from the body; decreased sodium levels lead to diminished circulating blood volume that in turn contributes to low blood pressure (known as “hypotension”), dehydration, weakness, and depression; increased levels of potassium in the blood (known as “hyperkalemia”) may result in heart-muscle toxicity

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Great Danes, rottweilers, Portuguese water dogs, standard poodles, West Highland white terriers, and soft-coated wheaten terriers have increased risk as compared to other dog breeds
- No breed predilection in cats

Mean Age and Range

- Dogs—range, less than 1 year to greater than 12 years of age; median, 4 years of age
- Cats—range, 1 to 9 years of age; most are middle-aged

Predominant Sex

- Female dogs are more likely to have hypoadrenocorticism than male dogs
- No predominant sex in cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary from mild and few in some patients with long-term (chronic) low levels of steroids produced by the adrenal glands (hypoadrenocorticism) to severe and life-threatening disease in a sudden (acute) Addisonian crisis (condition in which patient is in shock and collapse, usually with low body temperature [known as “hypothermia”], weak pulse, and an unexpectedly slow heart rate)
- Dogs—sluggishness (lethargy); lack of appetite (anorexia); vomiting; weight loss; signs vary in intensity—they may increase and decrease over time (known as a “waxing and waning” course); diarrhea; shaking; increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)
- Dogs—depression; weakness; dehydration; collapse; low body temperature (known as “hypothermia”); black, tarry stools (melena); weak pulse; slow heart rate (known as “bradycardia”); painful abdomen; hair loss
- Cats—sluggishness (lethargy); lack of appetite (anorexia); vomiting; increased urination (polyuria) and increased thirst (polydipsia); weight loss
- Cats—dehydration; weakness; weak pulse; slow heart rate (bradycardia)

CAUSES

- Primary hypoadrenocorticism—unknown cause (so called “idiopathic disease”); immune-mediated disease; side effect of medication (mitotane) used to treat excessive production of steroids by the adrenal glands (condition known as “hyperadrenocorticism” or “Cushing’s disease”); cancer
- Secondary hypoadrenocorticism—side effect of medical treatment with steroids, when long-term steroid administration is discontinued; abnormalities in the pituitary gland; the “pituitary gland” is the master gland of the body—it is located at the base of the brain; it

controls many other glands in the body

RISK FACTORS

- Treatment for excessive production of steroids by the adrenal glands (hyperadrenocorticism or Cushing's disease)
- Long-term use of steroids in medical treatment



TREATMENT

HEALTH CARE

- A sudden (acute) Addisonian crisis (condition in which patient is in shock and collapse, usually with low body temperature [hypothermia], weak pulse, and an unexpectedly slow heart rate) is a medical emergency requiring intensive therapy
- Treat sudden (acute) Addisonian crisis with rapid correction of low blood volume (known as “hypovolemia”) using isotonic fluids (preferably 0.9% NaCl)
- Treatment of long-term (chronic) hypoadrenocorticism depends on severity of clinical signs; usually initial stabilization and therapy are conducted on an inpatient basis

ACTIVITY

- No alteration necessary

DIET

- No alteration necessary



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- In an Addisonian crisis (condition in which patient is in shock and collapse, usually with low body temperature [hypothermia], weak pulse, and an unexpectedly slow heart rate), administration of a rapidly acting glucocorticoid or steroid (such as dexamethasone sodium phosphate or prednisolone sodium succinate) by injection is indicated
- Fluid therapy with 0.9% NaCl as needed, based on the patient's hydration, volume status and blood pressure
- Long-term (chronic) primary hypoadrenocorticism—treat with adrenal hormone replacement medications (glucocorticoid replacement [prednisone] and mineralocorticoid replacement [desoxycorticosterone pivalate or “DOCP” or fludrocortisone acetate])
- DOCP usually is required at monthly intervals, a few patients need injections every 3 weeks, and rare patients need injections every 2 weeks
- Patients with confirmed secondary hypoadrenocorticism require only glucocorticoid or steroid supplementation (prednisone)



FOLLOW-UP CARE

PATIENT MONITORING

- After the first 2 injections of DOCP, ideally do blood work and measure serum electrolyte (especially sodium and potassium) levels at 2, 3, and 4 weeks to determine duration of effect; thereafter, check electrolyte levels at the time of injection for the next 3 to 6 months (and adjust the dosage of DOCP, if necessary) and then every 6 months
- Adjust the daily dose of fludrocortisone, based on serial blood work (serum electrolyte determinations); following initiation of therapy, check serum electrolyte levels weekly until they stabilize in the normal range; thereafter, check serum electrolyte concentrations and blood urea nitrogen or creatinine monthly for the first 3 to 6 months and then every 3 to 12 months

PREVENTIONS AND AVOIDANCE

- Continue adrenal hormone replacement therapy for the lifetime of the patient
- Increase the dosage of replacement glucocorticoids or steroids during periods of stress (such as travel, hospitalization, and surgery), as

directed by your pet's veterinarian

POSSIBLE COMPLICATIONS

- Increased urination (polyuria) and increased thirst (polydipsia) may occur from prednisone administration, necessitating decreasing or discontinuing the drug
- Increased urination (polyuria) and increased thirst (polydipsia) may occur from fludrocortisone administration, necessitating a change to DOCP therapy

EXPECTED COURSE AND PROGNOSIS

- Most patients carry a good to excellent prognosis following proper stabilization and treatment
- Patients with underlying tumors or cancer have less favorable prognoses



KEY POINTS

- Lifelong glucocorticoid and/or mineralocorticoid replacement therapy is required
- Increased dosages of replacement glucocorticoids or steroids are required during periods of stress (such as travel, hospitalization, and surgery), as directed by your pet's veterinarian

HYPOCALCEMIA

(LOW LEVELS OF CALCIUM IN THE BLOOD)



BASICS

OVERVIEW

- “Hypocalcemia” is a low total serum calcium concentration in the blood
- “Parathyroid hormone” (also known as “parathormone” or “PTH”) is produced by the parathyroid glands; it regulates calcium and phosphorus levels in the blood—it normally increases calcium levels by causing calcium to be reabsorbed from bone
- The “parathyroid glands” are small, hormone-secreting glands that are located on or near the thyroid glands; thus the name, as “para-” refers to “adjacent” or “alongside” and “thyroid” refers to the thyroid gland; the thyroid and parathyroid glands are located in the neck, near the windpipe or trachea

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs of underlying disease may be seen without clinical signs of low levels of calcium in the blood (hypocalcemia), because the latter do not occur until total serum calcium falls below 6.7 mg/dl
- Seizures
- Muscle trembling, twitching, or involuntary contractions of groups of muscle fibers (known as “fasciculations”)
- Wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”) or stiff gait
- Weakness
- Panting
- Facial rubbing
- Vomiting
- Lack of appetite (known as “anorexia”)
- Fever
- Cataracts in patients with low levels of parathyroid hormone (known as “hypoparathyroidism”)

CAUSES

- Low levels of parathyroid hormone produced by the parathyroid gland (known as “primary hypoparathyroidism”); parathyroid hormone regulates calcium levels in the blood—it normally increases calcium levels by causing calcium to be reabsorbed from bone; “primary hypoparathyroidism” refers to a condition in which the glands do not produce adequate amounts of parathyroid hormone, resulting in a decrease in calcium levels and an increase in phosphorus levels in the blood
- Low levels of parathyroid hormone (hypoparathyroidism) secondary to surgical removal of the thyroid glands (known as “thyroidectomy”) or other corrective treatments for excessive production of thyroid hormone (known as “hyperthyroidism”) and subsequent parathyroid gland damage
- Hypoparathyroidism secondary to ultrasound-guided parathyroid gland radiofrequency heat ablation (for treatment of hyperparathyroidism or parathyroid masses) and parathyroid gland damage
- Kidney failure
- Ethylene glycol (chemical in many types of antifreeze) toxicity
- Oxalate toxicity (possible cause includes eating plants [such as lilies, philodendron])
- Sudden (acute) inflammation of the pancreas (known as “pancreatitis”)
- Complication of pregnancy or nursing (known as “eclampsia”)
- Phosphate-containing enemas
- Nutritional secondary hyperparathyroidism, caused by diets that have too much phosphorus and/or too little calcium and vitamin D—it is a type of malnutrition
- Abnormal absorption of calcium from the intestines
- Low levels of magnesium in the blood (known as “hypomagnesemia”)
- Citrate toxicity

- Rickets (disease caused by vitamin D deficiency)

RISK FACTORS

- Complication of pregnancy or nursing (eclampsia)—usually seen in small-breed dogs during the first 21 days of nursing a litter



TREATMENT

HEALTH CARE

- Inpatient treatment for patients with clinical signs of low levels of calcium in the blood (hypocalcemia), in which underlying disease requires support
- Emergency treatment usually is only needed for certain patients (such as those with primary hypoparathyroidism, hypoparathyroidism secondary to hyperthyroid or hyperparathyroid corrective procedures and subsequent parathyroid damage, complications of pregnancy or nursing [eclampsia], recent phosphate-containing enema administration, and citrate toxicity)
- Short-term and long-term treatment usually is needed only to treat primary hypoparathyroidism and complications of pregnancy or nursing (eclampsia)

ACTIVITY

- Depends on condition and underlying cause

DIET

- Diet change recommended in patients with nutritional secondary hyperparathyroidism, caused by diets that have too much phosphorus and/or too little calcium and vitamin D—it is a type of malnutrition



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Emergency Treatment

- Calcium gluconate 10% solution—administered slowly through a vein
- Calcium chloride 10% solution—also effective; administered slowly through a vein; extremely caustic if it gets outside of the vein and into tissues surrounding the vein; more potent than calcium gluconate
- If the patient has complications from nursing (eclampsia), may need to remove the puppies from the mother and hand-nurse until weaned

Short-term Treatment Immediately After Emergency Treatment

- Following emergency use of calcium gluconate 10% solution, relapse of clinical signs can be prevented by use of one of the following: constant-rate intravenous infusion; administration of calcium gluconate diluted in saline three to four times daily under the skin (subcutaneous administration)

Long-term Treatment of Hypocalcemia

- Vitamin D is needed indefinitely; dose as recommended by your pet's veterinarian
- Calcium supplements given by mouth; type and dose of calcium supplement as directed by your pet's veterinarian



FOLLOW-UP CARE

PATIENT MONITORING

- For patients requiring long-term treatment for low levels of calcium in the blood (hypocalcemia), blood work (serum calcium concentration) should be assessed in 4 to 7 days following initial treatment, then if patient has normal calcium levels, repeat blood work monthly for the first 6 months, then every 2 to 4 months; more frequent monitoring may be necessary if calcium levels are low
- Goal of treatment is to maintain serum calcium concentration between 8 and 10 mg/dl on blood work

POSSIBLE COMPLICATIONS

- Low levels of calcium in the blood (hypocalcemia)
- Excessive levels of calcium in the blood (known as “hypercalcemia”), which can lead to kidney failure

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause



KEY POINTS

- “Hypocalcemia” is a low total serum calcium concentration in the blood
- Signs of underlying disease may be seen without clinical signs of low levels of calcium in the blood (hypocalcemia), because the latter do not occur until total serum calcium falls below 6.7 mg/dl
- Goal of treatment is to maintain serum calcium concentration between 8 and 10 mg/dl on blood work

HYPOGLYCEMIA (LOW BLOOD SUGAR)



BASICS

OVERVIEW

- Abnormally low blood glucose (sugar) concentration

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog and cat

SIGNS/OBSERVED CHANGES in the ANIMAL

- Seizures
- Partial paralysis of the hindquarters or rear limbs (known as “posterior paresis”)
- Weakness
- Collapse
- Involuntary muscle twitches
- Abnormal behavior
- Sluggishness (lethargy) and depression
- Wobbly gait (known as “ataxia”)
- Increased appetite (known as “polyphagia”)
- Weight gain
- Increased urination (known as “polyuria” or “PU”) and increased thirst (known as “polydipsia” or “PD”)
- Exercise intolerance
- Some animals appear normal, aside from findings associated with underlying disease
- May have episodic signs

CAUSES

Endocrine

- Tumor involving cells of the pancreas that secrete the hormone, insulin (known as an “insulinoma”); excessive levels of insulin decrease the blood glucose levels
- Hormonal disturbances caused by cancer not involving the pancreas (known as “extrapancreatic paraneoplasia”)
- Overdose of prescribed insulin for treatment of diabetes (known as “iatrogenic insulin overdose”)
- Inadequate production of steroids by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”)

Hepatic Disease

- Portosystemic shunt (condition in which abnormal blood vessels allow blood to flow between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)
- Damage and scarring of the liver (known as “cirrhosis”)
- Severe inflammation of the liver (known as “hepatitis”)
- Glycogen-storage diseases—inherited disorders caused by a lack of normal enzymes to convert glycogen to glucose, resulting in greater than normal accumulation of glycogen in the liver; glycogen is the primary carbohydrate reserve in the body and is converted easily into glucose (sugar) under normal body conditions; it usually is found in the liver and other tissues in the body

Overuse of Glucose by the Body

- “Hunting-dog hypoglycemia” (condition seen in some hunting dogs, in which their blood glucose drops after one to two hours of strenuous exercise in the field)
- Pregnancy
- True increase in the number of red-blood cells in the body (known as “polycythemia”)
- Cancer
- Presence of pus-forming bacteria and their poisons in the blood or tissues (known as “sepsis”)

Reduced Intake/Underproduction of Glucose by the Body

- Young puppies and kittens

- Toy-breed dogs
- Severe malnutrition or starvation

RISK FACTORS

- Low intake of food for energy increases the likelihood of low blood sugar (hypoglycemia) in patients with conditions causing overuse of body glucose or underproduction of glucose by the body
- Fasting, excitement, exercise, and eating may or may not increase the risk of low blood sugar (hypoglycemic) episodes in patients with insulin-producing tumors (insulinomas)



TREATMENT

HEALTH CARE

- Treat animals with signs of low blood sugar (hypoglycemia) as inpatients
- Treat underlying disease
- If unable to eat, the veterinarian may start intravenous fluid therapy with 2.5% dextrose; if clinical signs persist, a 5% dextrose solution may be used

ACTIVITY

- Depends on underlying disease

DIET

- If able to eat (that is, the animal is responsive and is not vomiting), feeding should be part or all of initial treatment
- Hunting dog hypoglycemia—feed moderate meal of fat, protein, and complex carbohydrates a few hours before hunting; can feed snacks (such as dog biscuits) every 3 to 5 hours during the hunt
- Toy-breed hypoglycemia—increase frequency of feeding; feed several meals a day, as directed by your pet's veterinarian
- Puppy and kitten hypoglycemia—increase frequency of feeding (nursing or hand feeding)

SURGERY

- Surgery is indicated if a portosystemic shunt (condition in which abnormal blood vessels allow blood to flow between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver) or insulinoma (tumor involving cells of the pancreas that secrete the hormone, insulin) is the cause of hypoglycemia



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Emergency/Acute Treatment

- In hospital—administer 50% dextrose
- At home—do not attempt to administer medication by mouth during a seizure; seizures related to low blood sugar (known as “hypoglycemic seizures”) usually stop within 1 to 2 minutes; if a seizure is prolonged, recommend transportation to hospital; if a short seizure has ended or other signs of extremely low blood sugar (known as a “hypoglycemic crisis”) exist, recommend rubbing corn syrup or 50% dextrose on the tissues of the mouth, lining the cheek, and then followed by giving the same solution by mouth once the patient can swallow; then seek immediate veterinary medical attention
- Initiate frequent feeding of a diet low in simple sugars or, if patient is unable to eat, intravenous fluid therapy with 2.5% dextrose



FOLLOW-UP CARE

PATIENT MONITORING

- At home—for return or progression of clinical signs of low blood sugar (hypoglycemia); assess glucose levels on blood tests, if signs recur
- Single, intermittent serum glucose determinations may not truly reflect the effect of different foods on blood glucose (sugar) levels (known as “glycemic status”) of the patient
- Other monitoring is based on the underlying disease

PREVENTIONS AND AVOIDANCE

- Hunting dog hypoglycemia—feed moderate meal of fat, protein, and complex carbohydrates a few hours before hunting; can feed snacks (such as dog biscuits) every 3 to 5 hours during the hunt
- Toy-breed hypoglycemia—increase frequency of feeding; feed several meals a day, as directed by your pet’s veterinarian
- Puppy and kitten hypoglycemia—increase frequency of feeding (nursing or hand feeding)

POSSIBLE COMPLICATIONS

- Recurrent, progressive episodes of low blood sugar (hypoglycemia)
- Seizures

EXPECTED COURSE AND PROGNOSIS

- Depends on underlying disease



KEY POINTS

- Abnormally low blood glucose (sugar) concentration
- Treat animals with signs of low blood sugar (hypoglycemia) as inpatients
- Treat underlying disease
- Low intake of food for energy increases the likelihood of low blood sugar (hypoglycemia) in patients with conditions causing overuse of body glucose or underproduction
- Fasting, excitement, exercise, and eating may or may not increase the risk of low blood sugar (hypoglycemic) episodes in patients with insulin-producing tumors (insulinoma)

HYPOPARATHYROIDISM

(INADEQUATE PRODUCTION OF PARATHYROID HORMONE)



BASICS

OVERVIEW

- Absolute or relative deficiency of parathyroid hormone secretion leading to low levels of calcium in the blood (hypocalcemia)
- Parathyroid hormone regulates calcium and phosphorus levels in the blood—it normally increases calcium levels by causing calcium to be reabsorbed from bone
- The “parathyroid glands” are small, hormone-secreting glands that are located on or near the thyroid glands; thus the name, as “para-” refers to “adjacent” or “alongside” and “thyroid” refers to the thyroid gland; the thyroid and parathyroid glands are located in the neck, near the windpipe or trachea

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Toy poodle, miniature schnauzer, German shepherd dog, Labrador retriever, and terrier breeds
- Mixed-breed cats

Mean Age and Range

- Dogs—mean age, 4.8 years; range, 6 weeks to 13 years of age
- Cats—secondary to surgical removal of the thyroid glands (known as “thyroidectomy”) for treatment of excessive levels of thyroid hormone (known as “hyperthyroidism”): mean age, 12 to 13 years; range, 4 to 22 years of age; spontaneous hypoparathyroidism: mean age, 2.25 years; range, 6 months to 7 years of age

Predominant Sex

- Dogs—female (60%)
- Cats—male (64%)

SIGNS/OBSERVED CHANGES in the ANIMAL

Dogs

- Seizures
- Tense, splinted abdomen
- Wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”) or stiff gait
- Fever
- Facial rubbing
- Muscle trembling, twitching, or involuntary contractions of groups of muscle fibers (known as “fasciculations”)
- Growling
- Panting
- Cataracts
- Weakness
- Increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)
- Vomiting
- Lack of appetite (known as “anorexia”)
- May have normal physical examination results

Cats

- Sluggishness (lethargy), lack of appetite (anorexia), and depression
- Seizures
- Muscle trembling, twitching, or involuntary contractions of groups of muscle fibers (fasciculations)
- Panting
- Cataracts

- Slow heart rate (known as “bradycardia”)
- Fever
- Low body temperature (known as “hypothermia”)

CAUSES

- Dogs—most commonly of unknown cause (so called “idiopathic” disease) or immune-mediated inflammation of the parathyroid gland (known as “parathyroiditis”)
- Cats—most commonly secondary to damaged or removed parathyroid glands during surgical removal of the thyroid glands (thyroidectomy) for treatment of excessive levels of thyroid hormone (hyperthyroidism); decrease in tissue of parathyroid glands of unknown cause (so called “idiopathic parathyroid gland atrophy”) and immune-mediated inflammation of the parathyroid glands (parathyroiditis) also seen

RISK FACTORS

- Dogs—no risk factors identified
- Cats—surgical removal of the thyroid glands (thyroidectomy) for treatment of excessive levels of thyroid hormone (hyperthyroidism)



TREATMENT

HEALTH CARE

- Hospitalize for medical management of low levels of calcium in the blood (hypocalcemia) until clinical signs of hypocalcemia are controlled and serum calcium concentration is greater than 7.0 mg/dl on blood work
- Inpatient treatment for patients with clinical signs of low levels of calcium in the blood (hypocalcemia), in which underlying disease requires support
- Emergency treatment usually is only needed for certain patients (such as those with primary hypoparathyroidism or hypoparathyroidism secondary to procedures to correct excessive levels of thyroid hormone [hyperthyroidism] or excessive levels of parathyroid hormone [hyperparathyroidism] and parathyroid gland damage)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Emergency Treatment

- Calcium gluconate 10% solution—administered slowly through a vein
- Calcium chloride 10% solution—also effective; administered slowly through a vein; extremely caustic if it gets outside of the vein and into tissues surrounding the vein; more potent than calcium gluconate

Short-term Treatment Immediately After Emergency Treatment

- Following emergency use of calcium gluconate 10% solution, relapse of clinical signs can be prevented by use of one of the following: constant-rate intravenous infusion; administration of calcium gluconate diluted in saline three to four times daily under the skin (subcutaneous administration)

Long-term Treatment of Hypocalcemia

- Vitamin D is needed indefinitely; dose as recommended by your pet’s veterinarian
- Calcium supplements given by mouth; type and dose of calcium supplement as directed by your pet’s veterinarian



FOLLOW-UP CARE

PATIENT MONITORING

- Low levels of calcium in the blood (hypocalcemia) and excessive levels of calcium in the blood (hypercalcemia) are both concerns with long-term management
- Once serum calcium is stable and normal, assess serum calcium concentration monthly for the first 6 months then every 2 to 4

months; goal is to maintain serum calcium between 8 and 10 mg/dl on blood work

POSSIBLE COMPLICATIONS

- Low levels of calcium in the blood (hypocalcemia)
- Excessive levels of calcium in the blood (known as “hypercalcemia”), which can lead to kidney failure

EXPECTED COURSE AND PROGNOSIS

- With close monitoring of serum calcium and client dedication, the prognosis for long-term survival is excellent
- Adjustments in vitamin D and oral calcium administration can be expected during the course of management, especially during the initial 2 to 6 months of treatment
- Cats with low levels of parathyroid hormone (hypoparathyroidism) secondary to surgical removal of the thyroid glands (thyroidectomy) for excessive production of thyroid hormone (hyperthyroidism) and subsequent parathyroid gland damage usually require only transient treatment because they typically regain normal parathyroid gland function within 4 to 6 months following surgery and often within 2 to 3 weeks following surgery

HYPOTHERMIA (LOW BODY TEMPERATURE)



BASICS

OVERVIEW

- Body temperature below normal in an animal that normally maintains a relatively constant body temperature (known as a “homeothermic” or “warm-blooded” animal)
- Mild hypothermia—body temperature of 90° to 99° F (32° to 35° C)
- Moderate hypothermia—body temperature of 82° to 90° F (28° to 32° C)
- Severe hypothermia—any body temperature less than 82° F (28° C)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- More common in small breeds with increased likelihood of surface heat loss

Mean Age and Range

- More common in newborns and old animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Known prolonged exposure to cold ambient temperatures
- Possibly history of disappearance from home or of trauma
- Cold, unresponsive animal

Mild Hypothermia (Body Temperature of 90° to 99° F; 32° to 35° C)

- Mental depression
- Sluggishness (lethargy)
- Weakness
- Shivering

Moderate Hypothermia (Body Temperature of 82° to 90° F; 28° to 32° C)

- Muscle stiffness
- Slow heart rate (known as “bradycardia”)
- Low blood pressure (known as “hypotension”)
- Reduced breathing rate and depth
- Stupor/dullness

Severe Hypothermia (Body Temperature Less Than 82° F; 28° C)

- Unable to hear heart sounds
- Difficulty breathing
- Coma
- Fixed and dilated pupils

CAUSES

- Cold ambient temperature
- Impaired ability to regulate body temperature (such as in newborns, older animals, animals with low levels of thyroid hormone [known as “hypothyroidism”])
- Impaired behavioral responses—as seen in newborns or sick, debilitated, or injured animals
- Surface heat loss—as in newborns and small animals
- Inadequate heat generation—as in newborns and animals with extreme weight loss and muscle wasting

RISK FACTORS

- Low levels of thyroid hormone (hypothyroidism)
- Disease of the hypothalamus, the part of the brain that regulates appetite and body temperature

- Very young or old age
- Low body fat
- Anesthesia and surgery



TREATMENT

HEALTH CARE

- Treat most as inpatients until normal body temperature is reached
- Minimize movement to prevent lethal irregular heart beats (known as “cardiac arrhythmias”), especially in patients with severely low body temperature (severe hypothermia)
- Anticipate further drop in body temperature during initial rewarming
- Support vital organ systems, rewarm the patient, and prevent further heat loss
- Oxygen supplementation and breathing support may be necessary
- Mild hypothermia—use passive rewarming techniques, including thermal insulation with blankets
- Moderate hypothermia—use active external rewarming with heat sources (such as heating pads and radiant heat); apply heat to the trunk to rewarm the body’s “core;” provide a protective layer between the heat source and the patient’s skin
- Severe hypothermia—use core rewarming techniques, including warm water gastric and peritoneal lavage, warm water enemas, warm intravenous (IV) fluid administration, and airway rewarming (using warmed air)

ACTIVITY

- Minimize movement, especially in patients with severely low body temperature (severe hypothermia)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Oxygen supplementation may be provided via a face mask or endotracheal tube
- Blood volume support—essential; administer fluids intravenously
- Fluid solutions should be warm to prevent additional heat loss
- Fluid supplementation with dextrose may be helpful



FOLLOW-UP CARE

PATIENT MONITORING

- Core body temperature during rewarming
- Monitor electrocardiogram (“ECG,” a recording of the electrical activity of the heart) and blood pressure to assess status of circulatory system during rewarming
- Observe for development of frostbite

PREVENTIONS AND AVOIDANCE

- Avoid prolonged exposure to cold, especially with at-risk animals (such as small animals, older animals)
- Warm patient and monitor body temperature in anesthetized animals

POSSIBLE COMPLICATIONS

- Further drop in body temperature may occur during rewarming
- Return of cool blood to the heart may lead to irregular heart beats (cardiac arrhythmias)

- Severely low body temperature (severe hypothermia) may cause the heart to stop beating (known as “cardiac arrest”)

EXPECTED COURSE AND PROGNOSIS

- Varies with severity of low body temperature (hypothermia), underlying cause, and patient health status prior to hypothermic episode



KEY POINTS

- Avoid prolonged exposure to cold, especially with at-risk animals (such as small animals, older animals)
- Warm patient and monitor body temperature in anesthetized animals

HYPOTHYROIDISM

(LOW LEVELS OF THYROID HORMONE)



BASICS

OVERVIEW

- Clinical condition that results from inadequate production and release of thyroid hormone by the thyroid gland
- Characterized by a generalized decrease in metabolism

GENETICS

- No known genetic basis for the inheritance of primary hypothyroidism in dogs
- Familial (runs in certain families or lines of animals) inflammation of the thyroid gland characterized by the presence of lymphocytes (condition known as “lymphocytic thyroiditis”) has been reported in individual colonies of borzois, beagles, and Great Danes; “lymphocytes” are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and rarely cats

Breed Predilection

- Primary acquired (condition that develops sometime later in life/after birth) hypothyroidism is more common in medium- to large-sized dogs
- Breeds reported to have increased likelihood of developing primary acquired hypothyroidism as compared to other dog breeds include the golden retriever, Doberman pinscher, Irish setter, Great Dane, Airedale terrier, Old English sheepdog, dachshund, miniature schnauzer, cocker spaniel, poodle, and boxer

Mean Age and Range

- Most common in middle-aged dogs (4 to 10 years of age)

Predominant Sex

- No definitive predominant sex has been identified in affected dogs; however, castrated male dogs and spayed female dogs appear to be at increased risk of developing hypothyroidism

SIGNS/OBSERVED CHANGES in the ANIMAL

- Most common signs—sluggishness (lethargy); inactivity; mental dullness; weight gain; hair loss or excessive shedding; lack of hair regrowth following clipping; dry or lusterless hair coat; excessive scaling (accumulations of surface skin cells, such as seen in dandruff); darkened skin (known as “hyperpigmentation”); recurrent skin infections; and cold intolerance
- Uncommon signs—generalized weakness, incoordination, head tilt, facial paralysis, seizures, and infertility
- Clinical signs develop slowly and are progressive

Dogs

Skin Abnormalities—Very Common

- Symmetrical loss of hair on both sides of the trunk (known as “bilaterally symmetrical truncal alopecia”) that spares the head and extremities—common
- Hair loss (known as “alopecia”) is usually non-itchy, unless a secondary bacterial infection of the skin (known as a “secondary pyoderma”) or other itchy inflammation of the skin (known as “pruritic dermatitis”) also is present
- Hairs are removed from the hair follicles easily
- Hair loss (alopecia) occurs in areas of friction
- Hair loss (alopecia) often initially involves the flank area, base of the ears, tail (rat tail) and friction areas (such as under the front legs, lower chest, abdomen and neck, and under the collar)
- Early in the disease course, hair loss (alopecia) may be in multiple locations and not symmetrical; lesions may have irregular margins
- Darkened skin (hyperpigmentation) and increased thickness of the skin are common, particularly in friction areas
- Excessive scaling of the skin (known as “seborrhea”)—common; can be generalized, in multiple locations, or localized
- Dull, dry coat
- Secondary superficial bacterial infection of the skin (pyoderma) occurs occasionally; deep pyoderma is less common
- Accumulation of mucopolysaccharides in the skin can lead to nonpitting edema (known as “myxedema”), particularly in the facial area; this produces the classic “tragic” expression associated with hypothyroidism
- Inflammation of the outer ear (known as “otitis externa”) may be seen

General/Metabolic Abnormalities—Very Common

- Sluggishness (lethargy), mental dullness
- Weight gain
- Mild decrease in body temperature (low body temperature known as “hypothermia”)

Reproductive Abnormalities

- Infertility and prolonged interval between heat or estrus cycles (known as “anestrus”) in females

- Inappropriate white discharge that looks like milk from the nipples (known as “galactorrhea”) in sexually intact bitches; a “bitch” is a female dog

Abnormalities Involving the Nervous System and/or Muscular System—Uncommon

- Generalized weakness; dogs may have a stiff, stilted gait
- Other nervous system findings may include decreased reflexes (known as “hyporeflexia”), head tilt, facial paralysis, and a wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)
- A secondary muscle disease (known as a “myopathy”) usually is present in dogs with hypothyroid-related disease of many nerves (known as a “polyneuropathy”)
- Some hypothyroid dogs develop generalized muscle disease (myopathy) without coexistent nervous system involvement; these dogs present for generalized weakness
- Seizures have been reported rarely in hypothyroid dogs with marked increase in levels of lipids (a group of compounds that contain fats or oils) in the blood (known as “hyperlipidemia”)
- Paralysis of the larynx or voice box (known as “laryngeal paralysis”); enlargement of the esophagus (the tube running from the throat to the stomach; condition known as “megaesophagus”); and Horner’s syndrome (condition in which one pupil is small or constricted, the eyelid droops, and the eyeball is withdrawn into the socket) have been associated with hypothyroidism, but it is unknown if hypothyroidism causes these signs

Abnormalities of the Eyes

- Lipid (compound that contains fats or oils) deposits in the cornea; the “cornea” is the clear outer layer of the front of the eye
- Lipemia retinalis (condition in which the blood vessels in the back of the eye [retina] appear pink rather than normal red; pink color is caused by whitish lipids mixing with the blood)

Cats

- Rare
- Unkempt appearance; matting of hair; non-itchy, excessively dry scaling of the skin (known as “seborrhea sicca”); hair loss of the ears (known as “pinnal alopecia”)
- Sluggishness (lethargy)
- Obesity

Congenital (Present at Birth) Hypothyroidism—Cretinism

- Mental dullness/retardation, sluggishness (lethargy), inactivity
- Disproportionate dwarfism (large, broad head with short neck and limbs); shortened lower jaw (mandible); protruding tongue; delayed eruption of deciduous or baby teeth
- Constipation/obstipation—particularly in cats
- Low body temperature (hypothermia)
- Retention of puppy coat, progressive hair loss on the sides of the trunk (truncal alopecia) in dogs

CAUSES

- Inflammation of the thyroid gland characterized by the presence of lymphocytes (lymphocytic thyroiditis)
- Wasting away or decrease in size of the cells in the thyroid for unknown cause (so called “idiopathic thyroid atrophy”)
- Congenital (present at birth) thyroid disease
- Disease of the pituitary gland; the “pituitary gland” is the master gland of the body—it is located at the base of the brain; it controls many other glands in the body
- Dietary iodine deficiency; iodine is necessary for production of thyroid hormone
- Cancer
- Secondary to medication or treatment (known as “iatrogenic disease”)

RISK FACTORS

- Neutering may slightly increase risk of developing primary hypothyroidism
- Surgical removal of the thyroid (known as “thyroidectomy”)



TREATMENT

HEALTH CARE

- Outpatient

DIET

- Reduced-fat diet until body weight is satisfactory and serum thyroid hormone (T_4) concentrations are normal



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a

particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Levothyroxine is the treatment of choice; it is a thyroid (T_4) replacement hormone; also known as “L-thyroxine”
- Adjust dosage on the basis of serum thyroid hormone (T_4) concentration from blood obtained after giving the thyroid replacement medication and clinical response to therapy; initially, use a veterinary name-brand product
- If the patient responds to therapy, once-daily therapy can be tried; however, some patients require medication every 12 hours
- Different brands of L-thyroxine frequently have different absorption from the intestines; the dosage may change if the brand is changed
- Therapy with synthetic liothyronine (T_3) is not indicated or recommended in the vast majority of hypothyroid dogs
- Liothyronine (T_3) therapy is indicated only if a dog fails to achieve a normal serum T_4 concentration following appropriate therapy with at least two different brands of L-thyroxine, which probably indicates a lack of intestinal absorption—liothyronine is absorbed almost completely from the gut



FOLLOW-UP CARE

PATIENT MONITORING

- Check serum thyroid hormone (T_4) levels after 1 month of therapy
- Determine peak serum thyroid hormone (T_4) concentrations 4 to 8 hours after L-thyroxine administration
- Serum thyroid hormone (T_4) concentrations should be in the normal range or mildly increased; keep peak serum T_4 concentrations at or below 5 $\mu\text{g}/\text{dl}$ (64 nmol/L)
- Patients on once-daily therapy that do not respond to therapy and have a normal or high peak T_4 concentration should have their pre-pill T_4 concentration (trough T_4) assessed; if the trough T_4 concentration is low, twice-daily therapy is indicated
- Following initial normalization of serum T_4 values, check them yearly, or sooner if clinical signs of hypothyroidism or thyrotoxicosis (in which animal has signs of excessive thyroid hormone in the body; signs may include nervousness, weight loss, hyperactivity, and increased appetite) develop
- Recheck serum thyroid hormone (T_4) concentrations 1 month after any change in dosage or brand of L-thyroxine being administered

PREVENTIONS AND AVOIDANCE

- Proper treatment prevents disease recurrence

POSSIBLE COMPLICATIONS

- Prolonged administration of a high dosage of L-thyroxine can cause excessive levels of thyroid hormone (known as “iatrogenic hyperthyroidism”)
- Clinical signs of thyrotoxicosis (excessive thyroid hormone in the body) include panting; increased appetite (known as “polyphagia”); weight loss; increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”); anxiety; and diarrhea

EXPECTED COURSE AND PROGNOSIS

- Dogs treated for primary hypothyroidism have an excellent prognosis; life expectancy is normal
- Mental alertness and activity levels usually increase within 1 to 2 weeks after initiation of therapy
- Skin abnormalities slowly resolve over 1 to 4 months, as do nervous system deficits that are secondary to hypothyroidism
- Reproductive abnormalities resolve more slowly
- If significant clinical improvement does not occur within 3 months of initiation of therapy, with serum T_4 levels in the normal range, the diagnosis of hypothyroidism may be incorrect
- Patients with hypothyroidism may have a poor prognosis, if condition is secondary to a tumor or destructive process affecting the pituitary gland or hypothalamus



KEY POINTS

- Dogs with primary hypothyroidism respond well to treatment with oral synthetic thyroid hormone (levothyroxine or L-thyroxine)
- The appropriate dosage for L-thyroxine varies between individuals because of differences in gastrointestinal absorption of the medication and hormone metabolism
- Treatment is lifelong
- Most clinical and laboratory abnormalities resolve over a few weeks to a few months
- Occasionally, skin abnormalities worsen transiently during the first month of therapy

YELLOWISH DISCOLORATION TO THE TISSUES OF THE BODY (JAUNDICE OR ICTERUS)



BASICS

OVERVIEW

- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)
- Serum total bilirubin concentration higher than normal levels, leading to the yellowish discoloration
- “Bilirubin” is a normal bile pigment formed from the breakdown of hemoglobin; “hemoglobin” is the compound in the red-blood cells that carries oxygen to the tissues of the body; the liver takes up the hemoglobin following normal or abnormal breakdown of red-blood cells and processes it to form bile (a fluid substance involved in digestion of fats), which provides a means of eliminating bilirubin from the body
- Bilirubin levels in the blood can increase because more bilirubin is being made by the liver—in this case, more red-blood cells are being broken down (known as “hemolysis”) than usual, so the result is increased formation of bilirubin; bilirubin levels also can increase if the flow of bile is blocked (known as “cholestasis”)—in this case, the bilirubin is not eliminated from the body at a normal rate
- The liver is the largest gland in the body; it has many functions, including production of bile; bile ducts begin within the liver itself as tiny channels to transport bile—the ducts join together to form larger bile ducts and finally enter the extrahepatic or common bile duct, which empties into the upper small intestine; the system of bile ducts is known as the “biliary tree”
- The gallbladder is the storage unit for bile; bile is stored until it is needed for fat digestion

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- All breeds affected
- Breed predisposition for familial (runs in certain families or lines of animals) liver disease—Doberman pinschers, Bedlington terriers, cocker spaniels, Dalmatians, Labrador retrievers

Mean Age and Range

- Most causes—diseases of adult animals
- Young, unvaccinated dogs—at risk for infectious canine hepatitis

Predominant Sex

- Adult female purebred dogs—at risk for accelerated destruction or removal of red-blood cells related to an immune response, in which the body produces antibodies against red-blood cells (known as “immune-mediated hemolytic anemia”)

SIGNS/OBSERVED CHANGES in the ANIMAL

Increased Formation of Bilirubin—Breakdown of Red-Blood Cells (Hemolysis)

- Sluggishness (lethargy), weakness
 - Lack of appetite (known as “anorexia”)
 - Yellowish discoloration to the gums and other tissues of the body (jaundice or icterus)
 - Pale gums and moist tissues of the body (known as “mucous membranes”)
 - Enlargement of the liver (known as “hepatomegaly”) and/or enlargement of the spleen (known as “splenomegaly”)
 - Bleeding tendencies; low platelet or thrombocyte count (known as “thrombocytopenia”); “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
 - Orange feces
 - Enlarged lymph nodes (known as “lymphadenopathy”)
 - Fever
 - “Gelatinous” feel to skin (due to disease of the blood vessels [known as “vasculopathy”])
 - History of recent blood transfusion
 - Severe trauma: bleeding into muscle or formation of a localized mass of blood in a tissue or organ (known as a “hematoma”)
- #### *Decreased Elimination of Bilirubin—Blockage of the Flow of Bile (Cholestasis)*
- Sluggishness (lethargy)

- Lack of appetite (anorexia)
- Yellowish discoloration to the gums and other tissues of the body (jaundice or icterus)
- Change in color of urine and feces
- Abdominal enlargement
- Vomiting
- Diarrhea
- Increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)
- Altered mentation: brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”)
- Weight loss
- Enlargement of the liver (hepatomegaly) and/or enlargement of the spleen (splenomegaly)
- Build-up of fluid in the abdomen (known as “ascites”); possible mass in the abdomen; painful abdomen
- Black, tarry stools due to the presence of digested blood (known as “melena”) or pale or grayish coloration to the stools (known as “acholic feces”), due to the lack of bile pigments that cause the normal brown color of bowel movements: indicate complete blockage of the extrahepatic or common bile duct (extrahepatic bile duct obstruction)
- Fever

CAUSES

Icterus or Jaundice Caused by Problems Before the Liver (known as “Prehepatic Icterus or Jaundice”)

- Disorders causing breakdown of red-blood cells (hemolysis), such as immune-mediated hemolysis—certain drugs (propylene glycol carriers in cats, trimethoprim sulfa); systemic lupus erythematosus (autoimmune disease in which body attacks its own skin and other organs); infectious disorders; toxins (such as zinc, onions, phenols); severe low levels of phosphates in the blood (known as “hypophosphatemia”)
- Incompatible blood transfusion
- Infections—feline leukemia virus (FeLV); *Mycoplasma haemofelis*; heartworm; *Babesia*; *Ehrlichia*; *Cytauxzoon*
- Large volume blood resorption—localized mass of blood in a tissue or organ (hematoma), blood in body cavities (such as bleeding secondary to cancer [hemangiosarcoma], use of a product to decrease blood clotting [warfarin])

Icterus or Jaundice Caused by Problems Involving the Liver (known as “Hepatic Icterus or Jaundice”)

- Long-term (chronic) inflammation of the liver of unknown cause (so called “idiopathic hepatitis”) or that runs in certain families or lines of animals (known as “familial hepatitis”)
- Adverse drug reactions—such as those that occur after the use of medications to control seizures (known as “anticonvulsants”); acetaminophen; trimethoprim-sulfa (an antibiotic); carprofen (a pain reliever or analgesic); medication to improve appetite and weight gain (stanazolol) in cats; tranquilizers (benzodiazepines) in cats
- Inflammation of the bile duct or biliary tree (known as “cholangitis”) and inflammation of the bile ducts and liver (known as “cholangiohepatitis”)
- Cancer—lymphoma; “lymphoma” is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process
- Progressive damage and scarring of the liver (known as “cirrhosis”) in dogs
- Disease in which fats and lipids (compounds that contain fats or oils) accumulate in the liver of cats (condition known as “feline hepatic lipidosis”)
- Massive death of liver tissue (known as “liver necrosis”), such as due to aflatoxin
- Generalized (systemic) illnesses with liver involvement—certain types of leptospirosis (dogs); fungal infection (histoplasmosis); feline infectious peritonitis (FIP); excess levels of thyroid hormone (known as “hyperthyroidism”) in cats; toxoplasmosis (cats)
- Generalized bacterial infection (known as “sepsis”)—originating anywhere in the body; bacterial products may impair liver processing of bilirubin

Icterus or Jaundice Caused by Problems After the Liver (known as “Posthepatic Icterus or Jaundice”)

- Transient or persistent mechanical interference with the excretion of bilirubin and other bile elements: (1) inflammation of the pancreas (known as “pancreatitis”)—usually transient; (2) cancer—bile duct, pancreas, upper small intestine (known as the “duodenum”); (3) blockage of the bile duct—presence of hard, solid material in the bile duct (known as “cholelithiasis”), sludged bile, liver flukes (cats), immune-mediated duct destruction; (4) ruptured biliary tree causing leakage of bile and resulting inflammation of the lining of the abdomen (known as “bile peritonitis”)

RISK FACTORS

- Young unvaccinated dogs—infectious disease
- Breed predisposition for familial (runs in certain families or lines of animals) liver disease—Doberman pinschers, Bedlington terriers, cocker spaniels, Dalmatians, Labrador retrievers
- Middle-aged, obese dogs—inflammation of the pancreas (pancreatitis)

- Obese cats that have lost their appetite (known as “anorexia”)—disease in which fats and lipids (compounds that contain fats or oils) accumulate in the liver (hepatic lipidosis)
- Drugs that are toxic to the liver
- Blunt abdominal trauma, long-term (chronic) biliary tract disease—leakage of bile and resulting inflammation of the lining of the abdomen (bile peritonitis)
- Low number of red-blood cells due to breakdown of the cells (known as “hemolytic anemia”)



TREATMENT

HEALTH CARE

- Depends on underlying cause
- Inpatient—for initial medical care

ACTIVITY

- Cage rest—to facilitate liver regeneration

DIET

- Diet—important for cases with yellowish discoloration of the tissues due to problems of the liver (hepatic icterus or jaundice) and problems after the liver (posthepatic icterus or jaundice); nutritionally balanced with maximum protein tolerated by patient; carbohydrate based (dogs) with restricted protein for pets with the brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”); restrict sodium if pet has fluid build-up in the abdomen (ascites)
- Vitamin supplementation—water-soluble vitamins (vitamin B complex, vitamin C) in all patients; vitamin K₁ for patients with bile-duct blockage or obstruction or diseases in which the flow of bile is decreased or stopped (cholestasis)

SURGERY

- Depends on cause
- Surgical biopsy may be necessary to determine diagnosis or to treat certain conditions (such as bile duct blockage)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Yellowish discoloration of the tissues due to problems before the liver (prehepatic icterus or jaundice)—eliminate inciting cause; whole blood transfusion for life-threatening low red-blood cell counts (anemia)
- Yellowish discoloration of the tissues due to problems of the liver (hepatic icterus or jaundice) and problems after the liver (posthepatic icterus or jaundice)—treat specific disorder defined by biopsy and bacterial cultures



FOLLOW-UP CARE

PATIENT MONITORING

- Yellowish discoloration of the tissues due to problems before the liver (prehepatic icterus or jaundice)—recheck packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood) as needed
- Yellowish discoloration of the tissues due to problems of the liver (hepatic icterus or jaundice) and problems after the liver (posthepatic icterus or jaundice)—recheck blood work (serum biochemical profile) as dictated by underlying disease; continue symptomatic and specific treatment

PREVENTIONS AND AVOIDANCE

- Depend on underlying cause

POSSIBLE COMPLICATIONS

- Depend on underlying cause
- Diseases causing yellowish discoloration of the body tissues (jaundice or icterus) may cause death

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause



KEY POINTS

- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)
- “Bilirubin” is a normal bile pigment formed from the breakdown of hemoglobin; “hemoglobin” is the compound in the red-blood cells that carries oxygen to the tissues of the body; the liver takes up the hemoglobin following normal or abnormal breakdown of red-blood cells and processes it to form bile (a fluid substance involved in digestion of fats), which provides a means of eliminating bilirubin from the body
- Bilirubin levels in the blood can increase because more bilirubin is being made by the liver—in this case, more red-blood cells are being broken down (known as “hemolysis”) than usual, so the result is increased formation of bilirubin; bilirubin levels also can increase if the flow of bile is blocked (known as “cholestasis”)—in this case, the bilirubin is not eliminated from the body at a normal rate

FECAL INCONTINENCE

(INVOLUNTARY PASSAGE OF FECES OR BOWEL MOVEMENTS)



BASICS

OVERVIEW

- Inability to retain feces or bowel movements, resulting in involuntary passage of fecal material

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Although any age animal may be affected, incidence increases in older patients

SIGNS/OBSERVED CHANGES in the ANIMAL

Reservoir Incontinence (inadequate fecal-holding capabilities of the colon and/or rectum)

- Urge to defecate; frequent, conscious defecation without dribbling of feces; defecation may be associated with straining (known as “tenesmus”); difficulty in defecation (known as “dyschezia”) or blood in the stool (known as “hematochezia”)
- May have sensitivity or pain in the anus or rectum when the veterinarian feels the lower bowel on physical examination; an anal or rectal mass or thickening of the lining of the rectum may be detected; external anal sphincter (ring of muscle that opens and closes the opening of the anus) and anal reflex are normal

Non-Nervous System-Related Anal Sphincter Incontinence (the anal sphincter is the ring of muscle that opens and closes the opening of the anus)

- May include evidence of trauma to the skin and/or tissues surrounding the anus (known as “perineal tissues”) or deep infection involving the skin surrounding the anus, with extensive tracts (known as “perianal fistulas”); the anal reflex is present, but the external anal sphincter (ring of muscle that opens and closes the opening of the anus) may not close completely, if the sphincter has been disrupted structurally

Nervous System-Related Anal Sphincter Incontinence (the anal sphincter is the ring of muscle that opens and closes the opening of the anus)

- Involuntary expulsion or dribbling of bowel movement, especially during excitement, barking or coughing
- May include loss of tone to the external anal sphincter (ring of muscle that opens and closes the opening of the anus), but anal tone is a poor indicator of anal sphincter function; the anal reflex is absent or diminished
- Presence of urinary incontinence in addition to fecal incontinence suggests nervous system-related anal sphincter incontinence
- Additional findings suggesting lumbosacral spinal-cord disease include loss of voluntary movement and tone to the tail; pain in the area of the spine near the tail (known as “lumbosacral pain”); partial or complete paralysis, characterized by flabby muscles (lack of muscle tone), of the hindquarters and rear legs (known as “flaccid posterior paresis or paralysis”); and diminished response to nervous system testing of the reflexes to the rear legs

CAUSES

Reservoir Incontinence (inadequate fecal-holding capabilities of the colon and/or rectum)

- Diseases of the colon (large bowel) and/or rectum—inflammation of the colon (known as “colitis”); irritable bowel syndrome; cancer
- Diarrhea—large volumes of feces from any cause can overwhelm the absorptive and storage capacity of the colon

Non-Nervous System-Related Anal Sphincter Incontinence (the anal sphincter is the ring of muscle that opens and closes the opening of the anus)

- Traumatic injuries to the anus—bite wounds, lacerations, or gunshot wounds
- Disruption of the external anal sphincter and anal muscles during surgery of the anus and/or rectum
- Deep infection involving the skin surrounding the anus, with extensive tracts (perianal fistulas)

Nervous System-Related Anal Sphincter Incontinence (the anal sphincter is the ring of muscle that opens and closes the opening of the anus)

- Central nervous system disease—degenerative myelopathy, a disease of the spinal cord; birth defects (such as spinal dysraphism, spina bifida); trauma; intervertebral disk disease; cancer; inflammation of the spinal cord and its coverings (known as “meningomyelitis”) of various causes; blockage of blood vessels going to the spinal cord by pieces of fibrocartilage (known as “fibrocartilaginous embolism”) and other blood vessel disorders

- Cauda equina syndrome is a group of disorders in which the lumbosacral vertebral canal is narrowed, resulting in pressure on the nerve roots as they leave the spinal cord—intervertebral disk extrusion at the sixth-seventh lumbar (L₆-L₇) vertebral space or the seventh lumbar to the first sacral (L₇-S₁) vertebral space; formation of bony spurs around the edges of the vertebral endplates (known as “spondylosis deformans”); congenital (present at birth), narrowed vertebral canal (known as “congenital spinal canal stenosis”); lumbosacral instability; bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition is known as “diskospondylitis”); and cancer
- Nervous disorders involving the nerves outside of the central nervous system, such as the nerves to the anus—infectious; immune-mediated; drug-induced (such as vincristine sulfate, a chemotherapeutic drug); disorder of the autonomic nervous system (known as “dysautonomia”) and disorders/diseases of unknown cause (known as “idiopathic disorder or disease”)
- Muscular or nervous/muscular disorders
- Degeneration (aging)—multiple factors likely are involved, including loss of muscle mass (known as “muscular atrophy”) of the muscles involved in the control of defecation (known as “fecal continence”); weakness; degenerative nervous system disorders; and senility

RISK FACTORS

- Disease and/or surgery of the anus and/or rectum
- Nervous system disease



TREATMENT

HEALTH CARE

- If possible, identify and treat the underlying cause; fecal incontinence may resolve if the underlying cause is treated successfully
- Frequent warm water enemas may diminish the volume of feces in the colon and thus decrease the incidence of inappropriate defecation
- Environmental changes (such as making the pet an outside pet) may increase client satisfaction and thus avoid euthanasia of an otherwise healthy animal
- Reflex defecation can be induced sometimes in animals with hindquarter paralysis by applying a mild pinch on the toe on a pelvic limb or tail; similarly, applying a warm washcloth to the anus or perineum may stimulate defecation
- Improvement in signs may be achieved if specific therapy for perianal fistula, inflammatory bowel disease (IBD), or other reservoir or non-nervous system-related causes of incontinence can be given

ACTIVITY

- Depends on underlying cause

DIET

- Fecal volume can be reduced by feeding low-residue commercial diets or other foods, such as cottage cheese and rice and/or tofu
- Feed pet at established times to better control times needed to defecate

SURGERY

- Surgical reconstruction of lesions of the anus and/or rectum may improve fecal continence markedly in patients with non-nervous system-related anal sphincter (ring of muscle that opens and closes the opening of the anus) incontinence
- Various surgical procedures, involving the use of tissue or silicone slings, have met with variable success in treating nervous system-related anal sphincter incontinence in dogs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Opiate intestinal motility-modifying drugs (such as diphenoxylate hydrochloride and loperamide hydrochloride) increase segmental contraction of the bowel and slow passage of fecal material, thus increasing the amount of water absorbed from the feces
- Anti-inflammatory agents (such as steroids and sulfasalazine) may benefit patients with suspected reservoir incontinence due to inflammatory bowel disease (IBD)

- No specific drugs have been shown to be effective in patients with nervous system-related anal sphincter incontinence



FOLLOW-UP CARE

PATIENT MONITORING

- If fecal incontinence is due to an underlying nervous system cause, use serial neurologic examinations to monitor patient progress
- Diagnostic procedures (such as X-rays, electromyography [studies evaluating the speed of conduction of impulses in nerves], cerebrospinal fluid analysis) also can be used to follow progress
- Check fecal consistency and volume and make sure the pet does not become constipated
- Adjust diet and motility-modifying drug dosages to find the appropriate therapy for each individual patient

POSSIBLE COMPLICATIONS

- One recent report indicated that 50% of pets with fecal incontinence were euthanized

EXPECTED COURSE AND PROGNOSIS

- Prognosis is poor if the underlying cause cannot be identified and successfully corrected; discuss the prognosis with the client early in the evaluation, to avoid unrealistic expectations
- Nervous system-related anal sphincter incontinence often is unresponsive to treatment, despite appropriate dietary, medical, and surgical treatment



KEY POINTS

- Inability to retain feces or bowel movements, resulting in involuntary passage of fecal material
- If possible, identify and treat the underlying cause; fecal incontinence may resolve if the underlying cause is treated successfully
- Fecal volume can be reduced by feeding low-residue commercial diets or other foods, such as cottage cheese and rice and/or tofu
- Feed pet at established times to better control times needed to defecate
- Prognosis is poor if the underlying cause cannot be identified and successfully corrected; discuss the prognosis with the client early in the evaluation, to avoid unrealistic expectations
- Nervous system-related anal sphincter incontinence often is unresponsive to treatment, despite appropriate dietary, medical, and surgical treatment

URINARY INCONTINENCE

(INVOLUNTARY PASSAGE OF URINE)



BASICS

OVERVIEW

- Loss of voluntary control of urination, usually observed as involuntary urine leakage while resting

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and (rarely) cats

Breed Predilections

- Medium- to large-breed dogs most often affected

Mean Age and Range

- Most common in middle-aged to old spayed female dogs; also observed in juvenile females and (rarely) cats or neutered male dogs
- Usually develops within 3 years of spay (ovariohysterectomy), but can begin at any time, especially in geriatric dogs

Predominant Sex

- May affect more than 20% of spayed female dogs, especially large-breed dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Urine dribbling without the pet's control
- Wet hair coat on lower abdomen, area between rear legs and onto rear legs; inflammation of skin (known as "dermatitis") in these areas; animal may lick affected areas
- Evidence of urine wet spots or puddles in bedding, where animal was sleeping or sitting, other locations in house
- May have signs of urinary tract infection (such as straining to urinate, blood in the urine, painful urination)
- Urine scald (skin condition that looks like a burn due to the irritation of urine on the skin)
- Inflammation of the skin and moist tissues of the vulva or the prepuce (around the penis)
- Leakage may be worse after the pet drinks lots of water or exercises strenuously

CAUSES

Nervous-System Disorders

- Disruption of nerves involved in storage of urine in the bladder or act of urination
- Lesions of the sacral spinal cord, such as a birth defects; cauda equina syndrome, a group of disorders in which the lumbosacral vertebral canal is narrowed, resulting in pressure on the nerve roots as they leave the spinal cord; lumbosacral intervertebral disk disease; or traumatic fractures or dislocation can result in a flabby, overdistended urinary bladder; urine retention and overflow incontinence develop
- Lesions in the brain may affect voluntary control of voiding, usually resulting in frequent, involuntary urination or in leakage of small volumes of urine

Urinary Bladder-Storage Disorders

- Poor accommodation of urine during storage or urinary bladder overactivity (so called "overactive bladder" or "detrusor instability") leads to frequent leakage of small amounts of urine
- Urinary tract infections; chronic inflammatory disorders, such as "chronic cystitis;" cancer involving the bladder; pressure on the bladder from masses or fat; and chronic partial obstruction of the urethra (the tube from the bladder to the outside, through which urine flows out of the body)
- Underdevelopment of the bladder (known as "congenital urinary bladder hypoplasia") may accompany other congenital (present at birth) developmental disorders of the urinary and reproductive tracts
- The detrusor muscle acts to squeeze the bladder to expel urine; disorders of this muscle (known as "idiopathic detrusor instability") has been associated with feline leukemia virus (FeLV) infection in cats and unknown causes in dogs

Urethral Disorders (the urethra is the tube from the bladder to the outside, through which urine flows out of the body)

- Intermittent urinary incontinence is observed if urethral closure provided by urethral smooth muscle, striated muscle, and connective tissue does not prevent leakage of urine during storage
- Examples—underdevelopment of the bladder (congenital urinary bladder hypoplasia); acquired (develops during life/after birth) urethral

incompetence (such as reproductive hormone–responsive urinary incontinence); urinary tract infection or inflammation; prostatic disease or prostatic surgery (male dogs)

Anatomic or Structural Disorders

- Developmental or acquired anatomic abnormalities that divert urine from normal storage mechanisms or interfere with urinary bladder or urethral function
- The ureters are the tubes from the kidneys to the bladder; during development, they may not attach to the bladder properly or may attach to reproductive organs instead; when this occurs, they are called “ectopic ureters” and one or both can terminate in the distal urethra, uterus, or vagina
- Patent urachal remnants divert urine outflow to the umbilicus
- Abnormalities of the vagina, bladder or urethra
- Intrapelvic bladder neck location may contribute to urine leakage due to urethral incompetence (the inability of the urethra to prevent urine flow)
- Conformation abnormalities of the vulva or tissues around the vulva may contribute to urine pooling and intermittent urine leakage

Urine Retention

- Overflow observed when pressure within the bladder exceeds the ability of the sphincter and urethra to prevent urine flow

Mixed Urinary Incontinence

- Mixed or multiple causes of urinary incontinence are observed in humans and probably occur in dogs and cats; combinations of urethral and bladder-storage disorders and combinations of anatomic or structural and functional disorders are most likely

RISK FACTORS

- Neutering is the main risk factor for urinary incontinence; however, many spayed female dogs do not develop incontinence
- Conformational characteristics (such as bladder neck position, urethral length, and vaginal abnormalities) may increase the risk of urinary incontinence in female dogs
- Obesity may increase the risk of urinary incontinence in spayed female dogs
- Other possible risk factors for urethral incompetence include breed, large body size, and early tail docking



TREATMENT

HEALTH CARE

- Your pet’s veterinarian will assess your pet for potential causes of urinary incontinence and contributing factors
- Most cases will be managed successfully with medication
- Some cases will require surgical correction of anatomic problems, or injection of bulking material (collagen) into the urethra to prevent leakage
- Usually as outpatient
- Address partial obstructive disorders and primary nervous system disorders specifically, if possible
- Identify urinary tract infection and treat appropriately

DIET

- Weight management to prevent or treat obesity may decrease risk of urinary incontinence

SURGERY

- Developmental urinary tract disorders (such as ectopic ureters and congenital urethral hypoplasia) often can be corrected surgically; functional abnormalities of urethral competence or urinary bladder storage function may accompany the anatomic or structural disorder and require medical treatment
- Surgical procedures and prosthetic sphincter implantation have been described for the treatment of incontinence that is poorly responsive to medical treatment



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Urethral Incompetence

- Manage with reproductive hormones (such as stilbestrol, diethylstilbestrol, conjugated estrogens, estriol, and testosterone) or α -adrenergic agents (such as phenylpropanolamine, phenylephrine, pseudoephedrine)
- Reproductive hormones and α -adrenergic agents can be administered in combination for a synergistic therapeutic effect
- Imipramine, a tricyclic antidepressant (TCA) with anticholinergic and α -agonist actions, provides an alternative method of treatment
- Deslorelin also has been used in cases that respond poorly to other medications

Detrusor Instability

- Manage with anticholinergic or anti-spasmodic agents (such as oxybutynin, propantheline, imipramine, flavoxate, and dicyclomine)
- Tolterodine, a commonly used agent in humans, has not been used widely in dogs

Prostatic Disease

- Antibiotics for the treatment of infection/inflammation of the prostate (known as “prostatitis”) or prostatic abscesses
- Drugs to treat benign prostatic hyperplasia (“enlarged prostate”) and to cause enlarged prostate gland to return toward normal size, such as finasteride



FOLLOW-UP CARE

PATIENT MONITORING

- Patients receiving α -adrenergic agents—observe during initial treatment period for adverse effects of the drug, including rapid heart rate, anxiety, and high blood pressure (hypertension)
- Patients receiving estrogen—initial, 1-month, and periodic complete blood counts
- Periodic urinalysis and urine bacterial culture (to check for possible urinary tract infection)
- Take your pet to the veterinarian if you notice an increase in frequency of urination, an increase in urine leaking, or blood in the urine
- Once a therapeutic effect has been observed, slowly reduce the dosage and frequency of administration of medications to the minimum required to control signs of incontinence; dosage and frequency of administration should be changed under the direction of your pet’s veterinarian
- Consider combination treatment (α -adrenergic agents with reproductive hormones or anticholinergic agents), deslorelin or surgical options, if poor response to single-agent medication

POSSIBLE COMPLICATIONS

- Recurrent and ascending urinary tract infection
- Urine scald (skin condition that looks like a burn due to the irritation of urine on the skin)
- Inflammation of the skin (dermatitis) and moist tissues of the vulva
- Inflammation of the skin (dermatitis) on lower abdomen, area between rear legs and rear legs
- Unmanageable incontinence

EXPECTED COURSE AND PROGNOSIS

- Most dogs with urinary incontinence will respond well to medications and have complete resolution of incontinence (with rare lapses)
- Some dogs will require an adjustment in medication dose or type over time (years)
- A smaller percentage of dogs will not respond well to medication and will require combination medications, collagen injections or surgical procedures to improve continence; these dogs usually will be “improved,” but not “cured”
- The prognosis for improvement is less optimistic for cats and for male dogs



KEY POINTS

- Loss of voluntary control of urination, usually observed as involuntary urine leakage
- Medium- to large-breed dogs most often affected
- Most common in middle-aged to old spayed female dogs
- Obesity may increase the risk of urinary incontinence in spayed female dogs
- Urinary tract infection is a possible complication of urinary incontinence

FEMALE INFERTILITY



BASICS

OVERVIEW

- Abnormal cycling, breeding failure, conception failure, or pregnancy loss
- The female dog is a “bitch;” the female cat is a “queen”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Dog breeds susceptible to low levels of thyroid hormone (known as “hypothyroidism”) may have a higher number of cases of female infertility than other breeds; breeds susceptible to hypothyroidism include the golden retriever, Doberman pinscher, dachshund, Irish setter, miniature schnauzer, Great Dane, poodle, and boxer

Mean Age and Range

- Animals of all ages; may be more common in old animals
- Dogs greater than 6 years of age—more likely to have underlying cystic endometrial hyperplasia, a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts; may be susceptible to uterine infection and failure of conception or implantation

Predominant Sex

- Female

SIGNS/OBSERVED CHANGES in the ANIMAL

- Failure to cycle
- Too frequent cycling
- Failure to breed with a male
- Normal act of breeding, with no subsequent pregnancy or delivery or birth (known as “parturition”)
- Infertility caused by hormonal disease—signs of skin abnormality (such as hair loss [known as “alopecia”]); generalized (systemic) signs of disease (such as increased thirst [known as “polydipsia”] and increased urination [known as “polyuria”])
- Known pregnancy (through physical examination, ultrasound examination) with no subsequent delivery or birth (parturition)

CAUSES

- Previous surgical removal of ovaries and uterus (known as an “ovariohysterectomy” or “spay”)—animals already mature when obtained and possibility of previous ovariohysterectomy

Dogs

- Insemination at the improper time in “heat” or “estrus”—most common
- Subclinical uterine infection
- Cystic endometrial hyperplasia, a condition in which the lining of the uterus thickens abnormally and contains fluid-filled sacs or cysts
- Male infertility factors
- Inadequate levels of thyroid hormone (hypothyroidism)
- Excessive levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Anatomic abnormality
- Chromosomal abnormality
- Abnormal ovarian function
- Infection with *Brucella canis*—always a possibility

Cats

- Similar causes to those of dogs
- Lack of sufficient stimulus during breeding to induce release of eggs (ovulation)
- Systemic viral or protozoal infection

RISK FACTORS

- *Brucella canis*—dogs
- Inadequate levels of thyroid hormone (hypothyroidism)
- Excessive levels of steroids either produced by the adrenal glands (hyperadrenocorticism or Cushing's disease) or the result of administration in medications (known as "iatrogenic hyperadrenocorticism" or "iatrogenic Cushing's disease")—dogs and cats
- Generalized (systemic) viral infection—canine herpesvirus; feline leukemia virus (FeLV); feline immunodeficiency virus (FIV)
- Generalized (systemic) protozoal infection, such as toxoplasmosis—dogs and cats
- Any long-term (chronic), debilitating disease condition—dogs and cats
- Congenital (present at birth) abnormalities of the vagina—dogs and cats; the "vagina" is the tubular passageway leading from the opening of the vulva (female external genitalia) to the cervix of the uterus



TREATMENT

HEALTH CARE

- Medication to decrease heat or estrus (known as "estrus suppression") for one to two heat or estrous cycles may benefit bitches with frequent cycling
- Cats—seasonal breeders; depend on photoperiod (that is, length of daylight); queens cycle when exposed to long day length, normally from late January to mid-October; induce year-round cycling by daily light exposure of 12 hours or more; for a noncycling cat during the breeding season, evaluate the queen's housing and exposure to light

SURGERY

- Surgery for vaginal abnormalities (dogs)—may improve breeding and vaginal delivery of puppies
- Surgical repair of a closed tubular genital tract (dogs and cats)—difficult procedure; prognosis for future fertility guarded
- Surgical drainage of ovarian cysts (dogs and cats)—effectiveness unknown
- One-sided removal of a cancerous ovary (dogs and cats)—future fertility depends on resumption of normal function of the remaining ovary and lack of spread of the cancer (known as "metastasis")



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics (dogs and cats)—for uterine infection; choice depends on bacterial culture and sensitivity test of the uterus or of vaginal discharge during heat or estrus
- L-thyroxine (thyroid hormone)—for inadequate levels of thyroid hormone (hypothyroidism)

Hormones to Stimulate the Ovaries (known as "Gonadotropin Therapy")

- For induction of release of eggs (ovulation)
- Gonadotropin-releasing hormone, which causes release of luteinizing hormone from the pituitary gland—"luteinizing hormone" is the hormone that causes growth and maturation of eggs in the ovary; or human chorionic gonadotropin, which has luteinizing-like activity
- Cats not adequately stimulated to ovulate at the time of breeding—gonadotropin-releasing hormone at time of breeding
- Ovarian cystic disease—dogs and cats: gonadotropin-releasing hormone or human chorionic gonadotropin; causes release of eggs (ovulation) or luteinization of cystic ovarian tissue
- Heat or estrous induction (dogs)—diethylstilbestrol until signs of early heat or estrus are induced; bromocriptine (not approved for veterinary use in the United States and Canada); cabergoline
- Medications to suppress heat or estrus (dogs)—megestrol acetate or mibolerone



FOLLOW-UP CARE

PATIENT MONITORING

- L-thyroxine (thyroid hormone) treatment in dogs—blood concentrations of thyroid hormones (known as “T₃” and “T₄”) rechecked after one month of supplementation to ensure adequate absorption of medication and resumption normal thyroid levels
- Ultrasound examination in dogs and cats—to definitively diagnose pregnancy; monitor gestation
- Progesterone assay in dogs and cats; “progesterone” is a female hormone, which supports and maintains the pregnancy

PREVENTIONS AND AVOIDANCE

- Inherited cause of female infertility (such as inadequate levels of thyroid hormone [hypothyroidism])—consider advisability of retaining the animal in the breeding program

POSSIBLE COMPLICATIONS

- Depend on underlying cause
- Bitches with a vaginal anatomic abnormality—persistent or recurrent urinary tract disease or inflammation of the vagina (known as “vaginitis;” the “vagina” is the tubular passageway leading from the opening of the vulva [external genitalia] to the cervix of the uterus)

EXPECTED COURSE AND PROGNOSIS

- Prognosis for future fertility—initially good, because the most common cause of female infertility is improper breeding management; prognosis worsens with other causes
- Pets with inadequate levels of thyroid hormone (hypothyroidism) treated with thyroid hormone—prognosis for future fertility guarded with return to normal thyroid hormone levels



KEY POINTS

- Abnormal cycling, breeding failure, conception failure, or pregnancy loss
- Most common cause of female infertility is improper breeding management
- Inherited cause of female infertility (such as inadequate levels of thyroid hormone [hypothyroidism])—consider advisability of retaining the animal in the breeding program

MALE INFERTILITY—DOGS



BASICS

OVERVIEW

- Diminished or absent fertility in the male dog; does not imply sterility
- Results from a wide range of problems that prevent delivery of sufficient number of sperm to fertilize eggs in the bitch
- The male dog is the “stud dog;” the female dog is a “bitch”
- The male reproductive tract consists of two testicles (normally located in the scrotum), the epididymides (where sperm are stored prior to ejaculation), the deferent ducts (also known as the “vas deferens,” which are continuations of the ducts of the epididymides, through which semen moves; the deferent ducts enter the prostate and open into the urethra), the spermatic cord, the prostate, the penis, and the urethra (the tube that runs from the bladder and through the penis; urine or semen pass through the urethra)

GENETICS

- Very few inherited causes of infertility in the stud dog have been substantiated
- Alpha-L-fucosidase deficiency—causes impaired sperm maturation in male dogs; a lysosomal storage disorder has been reported—“storage disorders or diseases” are inherited metabolic diseases in which harmful levels of materials accumulate in the body’s cells and tissues; autosomal recessive inheritance in English springer spaniels
- Lack or abnormal motility of sperm (known as “primary ciliary dyskinesia”)—congenital (present at birth) abnormality; absent, irregular, or asynchronous motility patterns of sperm (reported in several breeds; likely inherited as an autosomal recessive trait)
- Inadequate levels of thyroid hormone (known as “hypothyroidism”)—some thyroid disorders appear to be inherited in female dogs and have specific effects on the “heat” or “estrous” cycles; the effect of hypothyroidism on male fertility is less clearly defined and probably is minimal

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Relatively higher number of specific problems involving male infertility seen in certain breeds as compared to other breeds

Mean Age and Range

- Male infertility in dogs increases with age

Predominant Sex

- Males

SIGNS/OBSERVED CHANGES in the ANIMAL

- General complaint—no puppies produced; whelping (birth) rate less than 75% when bred with correct timing to fertile bitches; owner suspects male-dog infertility
- Lack of libido and abnormal breeding behavior
- Abnormalities of the male reproductive tract (such as masses or scarring of the sheath and penis, abnormal testicles, painful testicles, abnormal prostate)

CAUSES

- Incorrect timing of breeding—most common cause of male infertility

Congenital (present at birth) Causes

- Chromosomal abnormalities (XXY syndrome) and XX sex reversal (XX male syndrome)—males with underdeveloped testicles and no sperm production
- Defective development or absence of the cells that produce sperm (known as “germinal cell aplasia”)—biopsy reveals “Sertoli cell only” syndrome
- Defective development or absence of the epididymis or deferent ducts (vas deferens; condition known as “segmental aplasia of the epididymis or vas deferens”)—may involve one side (known as “unilateral”) or both sides (known as “bilateral”); causes either a low number of sperm in the ejaculate (known as “oligospermia”) or absence of sperm in the ejaculate (known as “azoospermia”)

Acquired (condition that develops sometime later in life/after birth) Causes

- Incomplete ejaculation—unfamiliar surroundings; slippery flooring; no bitch in “heat” or “estrus;” dominant owner or bitch present
- Obstruction of the epididymides or deferent ducts (vas deferens)—leads to absence of sperm (azoospermia) if both sides (bilateral) are

involved; sperm granuloma; spermatocele; sudden (acute) inflammation of the epididymis (known as “epididymitis” and/or the testicles (known as “orchitis”); long-term (chronic) inflammation leading to narrowing of the passageways for the sperm (narrowing known as “stenosis”); defective development or absence of the epididymis or deferent ducts (vas deferens; condition is segmental aplasia); tumor or cancer; previous surgical removal of a section of the deferent ducts or vas deferens (known as “vasectomy”)

- Inflammation or infection of the testes (orchitis)—especially cases caused by *Brucella canis* and *Escherichia coli*; requires prompt and aggressive treatment to prevent infertility
- Inadequate levels of thyroid hormone (hypothyroidism)—role unclear; evaluate thyroid function in dogs with poor semen quality—hypothyroidism in these dogs is extremely rare; may be associated with decreased libido
- Increased levels of prolactin in the blood (known as “hyperprolactinemia”)—role unclear; evaluate prolactin levels in cases with absence of sperm (azoospermia); “prolactin” is a hormone from the pituitary gland that stimulates milk production
- Excessive levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)—causes decrease in the size of the testicles (known as “testicular atrophy”); probably reversible
- Drugs—examples include medications to kill parasites (known as “parasiticides”); steroids; anabolic steroids; estrogens (female hormones); androgens (male hormones); progestogens (substances capable of producing the effects of the female hormone, progesterone); amphotericin B (used to treat fungal infections)—may interfere with or interrupt sperm production
- Environmental toxins—hormone-disrupting contaminants can affect the pituitary gland and the reproductive tract; effects in the dog are unknown
- Trauma, environmental damage, tumors or cancer of the testicles, generalized (systemic) disease, blockage of blood flow to the testicles, and heat stress—may cause transient infertility or sterility
- Prostatic disease—appears to reduce semen quality and libido
- Inbreeding—reduces fertility
- Inflammation of the testicles, characterized by the presence of lymphocytes (known as “lymphocytic orchitis”)—familial (runs in certain families or lines of animals) in some breeds (such as the beagle and borzoi); affected animals may be fertile when young; fertility declines at an accelerated rate with age; “lymphocytes” are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process
- Retrograde ejaculation—some retrograde or backward flow of the ejaculate into the bladder is normal; however, complete retrograde ejaculation will lead to infertility (rare)

RISK FACTORS

- Congenital (present at birth) disorders affecting reproductive function—not uncommon; tend to occur in selected breeds
- Stud dogs and bitches not tested for infectious disease (such as testing for *Brucella canis* and bacterial culture of the genital tract) before breeding; *Brucella canis* is a bacteria that causes reproductive problems in male and female dogs



TREATMENT

HEALTH CARE

- Supportive regimens—reducing environmental heat or other stress

ACTIVITY

- Restrict, if activity or use is thought to be producing increased body temperature (known as “hyperthermia”)
- No restriction for other causes of infertility

DIET

- Ensure adequate diet and mineral supplementation, as directed by your pet’s veterinarian
- Avoid supplementation of products that contain excessive or undefined amounts of steroid hormones (such as extracts of testicles, ovaries and adrenal glands)

SURGERY

- Surgical reattachment of blocked ducts has been successful in several cases



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Specific medications must be administered long enough and at a dosage that will ensure tissue penetration
- Antibiotics—chloramphenicol, trimethoprim-sulfa, erythromycin, and enrofloxacin; usually recommended for a minimum of 3 to 4 weeks to allow adequate and sustained antibiotic levels within the reproductive tract
- Pseudoephedrine—used with limited success in people with retrograde ejaculation (backward flow of the ejaculate into the bladder)



FOLLOW-UP CARE

PATIENT MONITORING

- Recheck at intervals that take into account the length of the sperm production cycle (60 days), but are frequent enough to allow detection of deteriorating condition

PREVENTIONS AND AVOIDANCE

- Avoid exposure to environmental temperature extremes (heat or cold)

EXPECTED COURSE AND PROGNOSIS

- Fair to good—cases of mistimed breeding; appropriately breed to fertile bitch
- Guarded—cases of confirmed infertility; less than 10% of infertile male dogs return to fertility after diagnosis and appropriate treatment



KEY POINTS

- Incorrect timing of breeding—most common cause of male infertility
- The testicles will require at least 60 days from correction of identified reversible causes to return to function
- Regularly have dog checked by your pet's veterinarian to ensure no worsening of the condition
- Not all causes may be reversible

INFLAMMATORY BOWEL DISEASE (IBD)



BASICS

OVERVIEW

- A group of gastrointestinal tract diseases that occur for unknown reason; characterized by inflammation of the lining of the intestines and accompanied by chronic gastrointestinal signs (such as vomiting, diarrhea, weight loss)
- Also known as “IBD”

GENETICS

- Susceptibility genes (like those seen in human inflammatory bowel disease [IBD]) have not been identified in dogs and cats
- Certain forms of IBD are more common in some breeds of dogs, suggesting a possible genetic component of the disease processes
- Association of inherited chromosome fragility with IBD suggested in humans
- Certain genes, which are important components of normal immune responses, may make an individual susceptible to the development of IBD

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Some dog breeds are more likely to develop inflammatory bowel disease (IBD) than other breeds; examples of specific diseases and the breeds they affect are immunoproliferative enteropathy of basenjis and Norwegian lundehunds; histiocytic colitis of French bulldogs and boxers; and gluten-sensitive enteropathy in Irish setters; an increased incidence of IBD also is seen in the German shepherd dog
- Siamese may be more likely to develop IBD than other cat breeds

Mean Age and Range

- Most common in middle-aged animals, although younger animals (less than 2 years of age) may be affected

SIGNS/OBSERVED CHANGES in the ANIMAL

- Dogs—chronic intermittent vomiting, large- and/or small-bowel diarrhea, and weight loss are common
- Cats—lack of appetite (known as “anorexia”) is most common, followed by weight loss, vomiting, and diarrhea
- Rumbling or gurgling noises in the gastrointestinal tract (known as “borborygmus”); presence of excessive gas in the stomach and intestines (known as “flatulence”); blood in the stool (known as “hematochezia”); abdominal pain; and stools with mucus are reported less commonly
- Animal may appear healthy or may be thin and depressed
- Poor haircoat is noted frequently
- Abdominal palpation may reveal painful, thickened bowel loops and enlarged mesenteric lymph nodes (especially in cats)

CAUSES

- Cause is unknown; most likely many factors lead to disease
- Cause likely involves complex interactions between the animal’s genetics; immune capabilities and response of the lining of the intestinal tract (known as “mucosal immunity”); and environmental (gastrointestinal bacteria) factors
- No convincing link definitively established with an infectious agent (such as virus or bacteria)
- *Giardia*, *Salmonella*, *Campylobacter*, and normal resident gastrointestinal bacteria have been implicated
- Meat proteins, food additives, artificial coloring, preservatives, milk proteins, and gluten (wheat) are proposed causative agents



TREATMENT

HEALTH CARE

- Outpatient, unless the patient is debilitated from dehydration; low protein in the blood (known as “hypoproteinemia”); or has extreme weight loss with muscle wasting (known as “cachexia”)

- If the patient is dehydrated or must not be given food or water by mouth because of vomiting, fluids (such as lactated Ringer's solution) should be administered
- If the animal has severely low levels of albumin in the blood (known as "severe hypoalbuminemia") due to loss of protein into the intestinal tract (known as "protein-losing enteropathy"), consider colloids; colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch

ACTIVITY

- No restrictions

DIET

- Dietary manipulation is important; use hypoallergenic diets exclusively as dietary factors likely contribute to disease
- Dietary requirements may be based on specific disease (for example, avoiding gluten or wheat in Irish setters with gluten-sensitive enteropathy)

SURGERY

- Unlike the situation with humans, no surgical procedures are available for relief of inflammatory bowel disease (IBD) in veterinary patients



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Depends on underlying cause
- Affected animals should be treated with drugs to suppress the immune response (known as "immunosuppressive drugs")



FOLLOW-UP CARE

PATIENT MONITORING

- Periodic evaluations may be necessary, until the patient's condition stabilizes
- No other follow-up may be required except yearly physical examinations and assessment during relapses

PREVENTIONS AND AVOIDANCE

- Depends on underlying cause
- Avoid foods, food ingredients, or artificial colorings that may contribute to intestinal inflammation

POSSIBLE COMPLICATIONS

- Dehydration; malnutrition; adverse drug reactions; low levels of protein in the blood (hypoproteinemia); low red-blood cell count (known as "anemia"); and diseases secondary to therapy
- Depends on underlying cause

EXPECTED COURSE AND PROGNOSIS

- Generally a good-to-excellent short-term prognosis



KEY POINTS

- Inflammatory bowel disease (IBD) is not cured, but is controllable in most cases
- Relapses are common
- Be patient during the various food and medication trials that often are necessary to get the disease under control
- Strictly adhere to diet recommended by your pet's veterinarian

LOCALIZED INFLAMMATORY MUSCLE DISORDERS OF THE CHEWING (MASTICATORY) MUSCLES AND THE MUSCLES OF THE EYE



BASICS

OVERVIEW

- “Masticatory muscles” are muscles involved with chewing; they include the temporalis muscle (located on the side of the head, inserting on to the lower jaw)—it raises the lower jaw (mandible) to close the mouth—and the masseter muscle (located at the cheek and angle of the lower jaw [mandible])—it raises the lower jaw (mandible) to close the mouth
- “Myopathy” is the general term for a disorder of muscle
- “Extraocular” is defined as being adjacent to the eyeball, but outside of the eyeball
- Masticatory myopathy—localized inflammation of the muscles of chewing (known as “mastication”) that involves the temporalis and masseter muscles and spares the muscles of the legs
- Extraocular myopathy—localized inflammation of the muscles adjacent to the eyeball only; spares the muscles of chewing (mastication) and muscles of the legs

GENETICS

- Unknown
- As with autoimmune diseases in general, the appropriate genetic background must exist
- Masticatory myopathy—muscle disorder seen in Cavalier King Charles spaniels; they have a familial (runs in certain families or lines of animals) form and can be affected at less than 6 months of age
- Extraocular myopathy—muscle disorder seen in golden retrievers, may have a genetic susceptibility

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs (common)
- Cats (rare)

Breed Predilections

- Various
- Masticatory myopathy—rottweiler, Doberman pinscher, Samoyed, and Cavalier King Charles spaniel develop severe forms
- Extraocular myopathy—golden retrievers

Mean Age and Range

- No obvious age susceptibility

Predominant Sex

- None obvious

SIGNS/OBSERVED CHANGES in the ANIMAL

- Masticatory myopathy—muscle disorder with signs usually related to abnormalities of jaw movement and jaw pain; usually requires laboratory testing to confirm diagnosis
- Masticatory myopathy—sudden (acute) or long-term (chronic) pain when opening the jaw; marked jaw pain with manipulation and/or persistent contraction of the masseter muscles with inability to open the mouth (known as “trismus” or “lockjaw”); inability to pick up a ball or get food into the mouth; sudden (acute) swelling of the muscles; progressive loss of muscle mass (known as “muscle atrophy”); protrusion of the eyes (known as “exophthalmos”) or if muscle atrophy is present, eyes may be sunken (known as “enophthalmos”); inability to open the jaw under anesthesia
- Extraocular myopathy—marked protrusion of both eyes (known as “bilateral exophthalmos”); impaired vision

CAUSES

- Immune-mediated disorder

RISK FACTORS

- Appropriate genetic background
- Possible previous bacterial or viral infection

- Vaccination may exacerbate active disease



TREATMENT

HEALTH CARE

- Outpatient
- Feeding tube—may be required with severe restrictions in jaw mobility; requires good hygiene and supportive care

DIET

- Masticatory myopathy—may require liquid food or gruel until jaw mobility is regained; may need a feeding tube to facilitate fluid and caloric intake

SURGERY

- Not indicated, except for placement of feeding tube (if necessary)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids—administered at levels to decrease the response of the immune system (known as “immunosuppressive dosages”), tapered as jaw mobility, swelling, and serum creatine kinase (a muscle enzyme) levels return to normal; maintained at lowest alternate-day dosage that prevents restricted jaw mobility; treat for a minimum of 6 months
- If the owner does not tolerate the side effects of steroids—institute a lower dose of steroids and combine with another drug (such as azathioprine, a chemotherapeutic drug that is used to decrease the immune response)



FOLLOW-UP CARE

PATIENT MONITORING

- Masticatory myopathy—return of jaw mobility and decreased serum creatine kinase (a muscle enzyme) concentration
- Extraocular myopathy—decreased swelling of extraocular muscles

POSSIBLE COMPLICATIONS

- Steroids—undesirable side effects (such as increased urination)
- Recurrence of clinical signs—treatment stopped too early
- Poor clinical response—inadequate dosages of steroids
- Inability to move the eyeball (known as “restrictive strabismus”)—extraocular myositis

EXPECTED COURSE AND PROGNOSIS

- Masticatory myopathy—jaw mobility should return to normal, unless the condition is long-term (chronic) and severe scarring (known as “fibrosis”) develops; good prognosis if treated early with adequate dosages of steroids
- Extraocular myopathy—good response to steroids; good prognosis unless long-term (chronic) with inability to move the eyeball (restrictive strabismus)



KEY POINTS

- Long-term steroid therapy may be required
- Residual loss of muscle mass (muscle atrophy) and restricted jaw movement may occur with long-term (chronic) disease of the chewing (masticatory) muscles

GENERALIZED INFLAMMATORY DISORDERS OF MUSCLES— POLYMYOSITIS AND DERMATOMYOSITIS



BASICS

OVERVIEW

- “Polymyositis” is defined as inflammation of several skeletal muscles at the same time; it is a condition in which skeletal muscles are damaged by inflammation dominated by the presence of lymphocytes (a type of white-blood cell) infiltrating the muscle tissue; specifically, it is an immune-mediated disease
- “Dermatomyositis” is defined as inflammation of several skeletal muscles (polymyositis) at the same time, in association with characteristic changes in the skin (thus, the “derma-” portion of the name)
- “Myopathy” is the general term for a disorder of muscle
- “Generalized” refers to involvement of the entire organ, body system, or animal

GENETICS

- Unknown
- As for autoimmune diseases in general, the appropriate genetic background must exist
- Familial (runs in certain families or lines of animals) form of autoimmune polymyositis in the Newfoundland
- Dermatomyositis—reported to have an autosomal dominant inheritance pattern in rough-coated collies and Shetland sheepdogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and rarely cats

Breed Predilections

- Polymyositis (inflammation of several muscles)—various breeds of dogs and cats may be affected; breed associated in the Newfoundland and boxer
- Dermatomyositis (inflammation of several muscles with associated skin changes)—reported in rough-coated collies, Shetland sheepdogs, and Australian cattle dogs

Mean Age and Range

- Polymyositis (inflammation of several muscles)—none obvious
- Dermatomyositis (inflammation of several muscles with associated skin changes)—3 to 5 months of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Polymyositis (inflammation of several muscles)—usually associated with a stiff-stilted gait, muscle pain, and/or muscle weakness; may see regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth) and megaesophagus (enlarged esophagus)
- Elevated serum creatine kinase (a muscle enzyme) levels—supports, but does not make, the diagnosis of inflammation of the muscles (known as “myositis”)—do not rule out polymyositis if creatine kinase is normal
- Stiff-stilted gait—sudden (acute) or long-term (chronic)
- Muscle swelling and/or loss of muscle mass (known as “muscular atrophy”)
- Variable muscle pain
- Generalized muscle weakness and exercise intolerance
- Regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth) or difficulty swallowing
- Generalized loss of muscle mass (muscle atrophy), including the muscles of chewing (mastication)
- Nervous system examination—not abnormal; may be a decreased gag reflex if muscles of the throat (pharynx) are affected
- Dermatomyositis (inflammation of several muscles with associated skin changes) in dogs—skin lesions

CAUSES

- Immune-mediated disease
- Infectious disease—*Toxoplasma gondii*; *Neospora canis*; tick-related diseases; bacterial infection uncommon
- Drug-induced disease
- Secondary or related to cancer (known as “paraneoplastic” or “preneoplastic” syndrome)

RISK FACTORS

- Appropriate genetic background
- Possibly previous bacterial or viral infection
- Tumor or cancer, possibly “occult;” occult is defined as “hidden”—it indicates that the primary tumor may not be identified
- Exposure to ticks



TREATMENT

HEALTH CARE

- Outpatient
- Supportive care—may be required to prevent skin wounds and decubital ulcers (“bed sores”) in nonambulatory, severely affected patients

ACTIVITY

- Should increase, along with muscle strength, as muscle inflammation decreases

DIET

- Megaesophagus (enlarged esophagus)—may require feeding from an elevated food bowl; elevate food and water bowls; try foods of different consistencies
- Severe regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth)—may need to place a feeding tube to maintain hydration and nutrition

SURGERY

- Muscle biopsy—needed to confirm the diagnosis
- Only if a tumor or cancer is the underlying cause of the muscle inflammation
- To place a feeding tube, in order to maintain hydration and nutrition



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids—administered at levels to decrease the response of the immune system (known as “immunosuppressive dosages”); usually result in clinical improvement of immune-mediated condition; decrease to the lowest alternate-day dosage that maintains normal creatine kinase (a muscle enzyme) and improved muscle strength and mobility; may require long-term therapy
- Identified infectious agent—initiate specific therapy for the infectious agent
- If side effects of steroids are not tolerated by the owner—institute a lower dose of steroids and combine with another drug (such as azathioprine, a chemotherapeutic drug used to decrease the immune response)



FOLLOW-UP CARE

PATIENT MONITORING

- Serum creatine kinase (a muscle enzyme)—periodic evaluation; if elevated, should decrease into the normal range
- Steroids—side effects

POSSIBLE COMPLICATIONS

- Steroids—undesirable side effects (such as increased urination)
- Recurrence of clinical signs—treatment stopped too early

- Poor clinical response—inadequate dosages of steroids

EXPECTED COURSE AND PROGNOSIS

- Immune-mediated condition—good to fair prognosis
- Disorder secondary or related to cancer (paraneoplastic disorder) associated with occult (hidden) tumor or cancer—guarded prognosis



KEY POINTS

- Long-term immunosuppressive therapy may be required for an immune-mediated condition
- Residual loss of muscle mass (muscle atrophy) and contracted muscles may occur with long-term (chronic) disease and extensive scarring (known as “fibrosis”)
- Suggest genetic counseling for familial (runs in certain families or lines of animals) disorders

INSULINOMA

(TUMOR INVOLVING CELLS OF THE PANCREAS THAT SECRETE THE HORMONE, INSULIN)



BASICS

OVERVIEW

- Pancreatic islet β -cell tumor that secretes an excess quantity of insulin, independent of glucose (sugar) levels in the blood, leading to low levels of glucose in the blood (known as “hypoglycemia”)
- The β -cells of the pancreas produce insulin, the hormone that regulates blood glucose (sugar) levels; under normal conditions, insulin responds to changes in blood glucose levels and keeps the blood glucose in a relatively narrow range—if the blood glucose levels increase over a certain level (generally around 110 mg/dl), insulin levels increase to push the blood glucose level down; if blood glucose levels fall below a certain level (generally about 60 mg/dl), insulin levels drop to allow the blood glucose to go up

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs—uncommon
- Cats—rare (4 reports)

Breed Predispositions

- Dogs—standard poodle, boxer, fox terrier, Irish setter, German shepherd dog, golden retriever, and collie
- Cats—none; possibly Siamese

Mean Age and Range

- Dogs—middle-aged to old; mean, 10.5 years of age; range, 3 to 14 years of age (rare in dogs less than 6 years of age)
- Cats—(4 cases); mean, 14.75 years of age; range, 12 to 17 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Episodic
- Signs may or may not be related to fasting, excitement, exercise, and eating
- Dogs usually demonstrate more than one clinical sign, and signs progress with time
- Dogs—seizures (generalized and focal) most common; also, partial paralysis of the hindquarters; weakness; collapse; involuntary muscle twitches; abnormal behavior; sluggishness (lethargy) and depression; wobbly gait (known as “ataxia”); increased appetite (known as “polyphagia”); weight gain; increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”); and exercise intolerance
- Cats—seizures; wobbly gait (ataxia); involuntary muscle twitches; weakness; sluggishness (lethargy) and depression; lack of appetite (known as “anorexia”); weight loss; and increased thirst (polydipsia)
- Physical examination usually normal
- Obesity in some dogs
- Rarely, nervous system disease involving several nerves (known as “polyneuropathy”) in dogs

CAUSES

- Most patients have malignant, insulin-producing cancer (known as a “carcinoma” or “adenocarcinoma”) of the pancreas; tumors that appear to be benign on microscopic sections usually metastasize later

RISK FACTORS

- Fasting, excitement, exercise, and eating may increase the risk of low blood sugar; behavior or signs related to low blood sugar are called “hypoglycemic episodes”



TREATMENT

HEALTH CARE

- Hospitalize for diagnostic workup and surgery, as well as for treatment of low blood sugar (hypoglycemia), if needed
- Treat as outpatient if the owner declines surgery, and if the patient does not have signs of low blood sugar (hypoglycemia)
- Administer 50% dextrose to control seizures and/or severe signs of low blood sugar
- Fluid therapy with 2.5% dextrose (increase to 5%, if needed to control clinical signs) should follow dextrose bolus; alternatively, if the patient can eat, frequent feedings of an appropriate diet may replace need for dextrose-containing fluids

ACTIVITY

- Restricted

DIET

- The first and most important aspect of management (with or without surgery)
- Feed 4 to 6 small meals a day
- Should be high in protein, fat, and complex carbohydrates and low in simple sugars; avoid semi-moist food

SURGERY

- Surgical removal of all or part of the pancreatic islet β -cell tumor confirms diagnosis, may improve survival time, potentially can provide prolonged remission, and may improve response to medical treatment; postoperative inflammation of the pancreas (known as “pancreatitis”) is possible



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Long-Term Therapy

- Steroids (such as prednisone)—initial medical treatment if diet alone is ineffective; begin with low dosage and gradually increase as signs of low blood sugar (hypoglycemia) recur
- Diazoxide (Proglycem®)—added after diet and steroids have proven ineffective
- Streptozocin—a nitrosourea that selectively kills pancreatic β -cells; administer with drugs to decrease vomiting (known as “antiemetics”), a side effect of the drug
- Glucagon—a gluconeogenic drug used to treat sudden low blood sugar that is poorly responsive to medical treatment
- Sandostatin® (octreotide or lantreotide)—a synthetic somatostatin analogue; prevents low blood sugar (hypoglycemia) in some dogs that are poorly responsive to conventional treatment; can be used with diet, steroids, and diazoxide; expensive



FOLLOW-UP CARE

PATIENT MONITORING

- At home: monitor for return or progression of clinical signs of low blood sugar (hypoglycemia)
- In-hospital blood glucose determinations—single, intermittent blood glucose determinations may not truly reflect control of blood sugar levels (known as “glycemic control”) of the patient because insulinomas occasionally respond to changing blood sugar levels
- Adjust medication on the basis of clinical signs and blood sugar levels and fructosamine (a particular protein found in blood used to monitor glycemic control) concentrations

POSSIBLE COMPLICATIONS

- Recurrent or progressive episodes of low blood sugar (hypoglycemia)

EXPECTED COURSE AND PROGNOSIS

- Likelihood of malignancy is high; metastasis is seen in 40% of patients at the time of surgery
- Dogs—mean survival time, about 16 to 19 months; range, 2 to 60 months; surgery improves survival time in some animals
- Cats—mean survival time, about 6.5 months; range, 0 to 18 months



KEY POINTS

- Be aware of signs of low blood sugar (hypoglycemia) and seek immediate veterinary medical attention if they occur
- Diet is the first and most important aspect of management (with or without surgery)
- Likelihood of malignancy is high; metastasis is seen in 40% of patients at the time of surgery

CERVICAL INTERVERTEBRAL DISK DISEASE IN DOGS



BASICS

OVERVIEW

- The spine is composed of multiple bones (vertebrae) with disks (intervertebral disks) located in between adjacent bones; the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral (located at the pelvis) and coccygeal (tail) vertebrae
- Each disk is composed of a central gel-like area, known as the “nucleus pulposus,” and an outer fibrous ring, known as the “annulus fibrosis”
- Degeneration of cervical intervertebral disks causes protrusion or extrusion of disk material into the spinal canal; the protruded or extruded disk material causes pressure on the spinal-cord itself (known as “spinal-cord compression” or “myelopathy”) and/or nerve-root compression (known as “radiculopathy”)
- Protrusion is defined as the disk bulging into the spinal canal with the fibrous ring of the disk being intact; extrusion is defined as the center or nucleus of the disk being forced out of its normal position into the spinal canal with the fibrous ring of the disk being ruptured
- Two types of protrusion/extrusion (“slipped disk”) have been reported in dogs: sudden (acute) disk herniation is Hansen type I and long-term (chronic) disk herniation is Hansen type II; Hansen type I involves degeneration of the center or nucleus of the disk with rupture of the fibrous ring and resulting movement of the center into the spinal cord (extrusion) while Hansen type II involves degeneration of the disk, followed by bulging of the disk into the spinal cord with the fibrous ring remaining intact (protrusion)

GENETICS

- Chondrodystrophoid breeds are dogs with shortened legs that are bowed to some degree; they include such breeds as the Pekingese and dachshund; the chondrodystrophoid breeds have accelerated disk degeneration as compared to other breeds
- Eighty percent of disk extrusion occurs in dachshunds, beagles, and poodles; incidence in the dachshund population is about 25%

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Hansen type I (sudden disk herniation)—dachshunds, poodles, beagles, Pekingese, cocker spaniels
- Hansen type II (slower, long-term disk herniation)—Doberman pinschers

Mean Age and Range

- Hansen type I—3 to 6 years of age
- Hansen type II—8 to 10 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Severity of signs is dependent on the rate and volume of disk protrusion or extrusion, and the diameter of the vertebral canal relative to spinal-cord size
- Neck pain
- Stiff, stilted gait, reluctance to move the head and neck
- Lowered head stance and muscle spasms of the head, neck, and shoulder
- 10% of affected patients are weak or partially paralyzed in all four limbs (known as “tetraparesis”)
- Neck pain—elicited upon flexion and extension of the neck, turning the neck from side to side or by deep palpation of the cervical muscles
- Forelimb lameness (such as knuckling, or limb held in partial flexion) as a result of pressure on the nerve roots as they exit the spinal cord helps localize the intervertebral disk lesion to the fourth to seventh cervical vertebrae (C₄-C₇)
- Partial paralysis (paresis) in both forelegs and rear legs may be present; the nervous system deficits/lameness also can be on only one side of the body (right side or left side) and involving the front leg and rear leg on that side
- Rear-leg partial paralysis (paresis) may be more severe than front-leg partial paralysis
- Rear-leg spinal reflexes may be normal to exaggerated
- Front-leg spinal reflexes may be normal to exaggerated when the intervertebral disk lesion is at the first to sixth cervical vertebrae (C₁

-C₆) or may be normal to decreased when at the sixth cervical vertebra to second thoracic vertebra (C₆-T₂)

- Bladder function may be normal or may be abnormal, characterized by a distended firm bladder that is difficult to empty manually (known as an “upper motor neuron bladder”)

CAUSES

- Hansen type I—early degeneration of the cervical intervertebral disk and subsequent disk mineralization
- Hansen type II—gradual degeneration of the cervical intervertebral disk

RISK FACTORS

- Obesity and breed predisposition



TREATMENT

HEALTH CARE

- Conservative management—indicated with gradual onset of clinical signs or clinical signs that are limited to an exaggerated response to painful stimuli (known as “hyperpathia”) or mild wobbly gait (known as “ataxia”)
- Surgical management—indicated for repeated episodes of neck pain; patients with severe neck pain and nervous system deficits; or patients that have not responded to conservative management
- Handling—minimal manipulation, avoid obtaining blood samples from the jugular vein
- Urination—monitor for complete emptying of the bladder; may need to express bladder manually or catheterize intermittently; some cases, may need indwelling urinary catheter
- Urine bacterial culture and sensitivity are recommended in all dogs undergoing spinal decompression surgery
- Defecation—monitor and adjust diet or give enemas as needed
- Recumbent patients—keep on a well-padded mat and turn every 4 hours, check for pressure or “bed” sores over bony prominences
- Physical therapy—institute hydrotherapy and passive range of motion of all joints as often as possible
- All patients should be fitted with a harness instead of a neck collar

ACTIVITY

- Minimal, no running or jumping.
- Walk using a harness, instead of a collar
- Conservative management patients strictly are confined to cage rest for 3 to 4 weeks
- Following surgery, leash-walk only for 4 to 6 weeks, then slowly re-introduce to full activity

DIET

- For obese patients, a reducing diet should be instituted

SURGERY

- The goal of surgery is to remove disk material from the spinal canal to decompress the spinal cord and/or affected nerve roots; surgery is known as “decompressive surgery” as it removes pressure from the spinal cord
- Surgery usually provides immediate pain relief and eventual return of normal motor function
- A “ventral cervical slot” is the most common surgical approach for the removal of disk material from the spinal canal
- Disk material that has moved into the intervertebral foramen often is removed via a lateral approach to the cervical spine
- Fenestration (surgical procedure in which disk material is removed from disks that are still in their normal location and are not extending into the spinal canal) without decompression is seldom recommended



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Low-dose steroid therapy may be beneficial, in order to decrease pain in animals that are being treated conservatively
- Steroids given to animals without simultaneous strict cage confinement could worsen disk extrusion by encouraging exercise

- Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used only if the animal is not currently, or recently, on steroids
- Muscle relaxants generally are unsuccessful when used alone



FOLLOW-UP CARE

PATIENT MONITORING

- Weekly evaluations until resolution of clinical signs

PREVENTIONS AND AVOIDANCE

- Inherent medical disorder in particular breeds; therefore, nothing actually prevents intervertebral disk disease
- Keeping patients at an ideal weight may help

POSSIBLE COMPLICATIONS

- Continued neck pain
- Deteriorating ability to stand and/or walk
- Dislocation of vertebrae after decompressive surgery

EXPECTED COURSE AND PROGNOSIS

- Prognosis depends on nervous system signs at time of presentation
- Generally favorable for most patients
- Many patients treated conservatively have recurrence of clinical signs



KEY POINTS

- For conservative management, emphasize strict cage confinement
- Weight loss, if the animal is obese
- Many patients treated conservatively have recurrence of clinical signs
- The goal of surgery is to remove disk material from the spinal canal to decompress the spinal cord and/or affected nerve roots; surgery is known as “decompressive surgery” as it removes pressure from the spinal cord
- Walk using a harness, instead of a collar

THORACOLUMBAR INTERVERTEBRAL DISK DISEASE



BASICS

OVERVIEW

- The spine is composed of multiple bones (vertebrae) with disks (intervertebral disks) located in between adjacent bones; the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral (located at the pelvis) and coccygeal (tail) vertebrae
- Each disk is composed of a central gel-like area, known as the “nucleus pulposus,” and an outer fibrous ring, known as the “annulus fibrosis”
- “Thoracolumbar intervertebral disk disease” refers to degenerative changes in the disks involving the thoracic and lumbar backbones (vertebrae) of the spine
- Degeneration of thoracolumbar intervertebral disks causes protrusion or extrusion of disk material into the spinal canal; the protruded or extruded disk material causes pressure on the spinal-cord itself (known as “spinal-cord compression” or “myelopathy”) and/or nerve-root compression (known as “radiculopathy”)
- Protrusion is defined as the disk bulging into the spinal canal with the fibrous ring of the disk being intact; extrusion is defined as the center or nucleus of the disk being forced out of its normal position into the spinal canal with the fibrous ring of the disk being ruptured
- Two types of protrusion/extrusion (“slipped disk”) have been reported in dogs: sudden (acute) disk herniation is Hansen type I and long-term (chronic) disk herniation is Hansen type II; Hansen type I involves degeneration of the center or nucleus of the disk with rupture of the fibrous ring and resulting movement of the center into the spinal cord (extrusion) while Hansen type II involves degeneration of the disk, followed by bulging of the disk into the spinal cord with the fibrous ring remaining intact (protrusion)
- Thoracolumbar disk disease comprises 85% of all disk herniations in dogs

GENETICS

- Chondrodystrophic breeds are dogs with shortened legs that are bowed to some degree; they include such breeds as the Pekingese, shih tzu, and dachshund; the chondrodystrophic breeds have accelerated disk degeneration as compared to other breeds; they have Hansen type I disease
- Larger breeds more commonly have Hansen type II disease

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Occasionally cats

Breed Predispositions

- Hansen type I disease—dachshunds; shih tzus, Lhaso apsos, Pekingese, cocker spaniels, Welsh corgis, and toy and miniature poodles
- Hansen type II disease—large-breed dogs, but may occur in any breed; cats

Mean Age and Range

- Hansen type I disease—dogs, 3 to 6 years of age
- Hansen type II disease—dogs, 8 to 10 years of age; cats, mean age of 10 years

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs depend on location and type of disk herniation (protrusion or extrusion), the velocity of disk contact with the spinal cord, and the amount and duration of spinal cord compression
- Onset may be very sudden (peracute) or sudden (acute) in chondrodystrophic dogs (Hansen type I disease), and may occur during vigorous activity
- Larger dogs or smaller dogs with Hansen type II disease have a more subtle onset, and tend to worsen with time
- Thoracolumbar pain is common in dogs; reluctance to move and hunched posture
- Often some degree of weakness of the hind limbs (known as “paraparesis”)
- Spinal reflexes in the hind limbs usually are exaggerated when the spinal cord lesion is located between the third thoracic (T₃) and third lumbar (L₃) vertebrae; reflexes are decreased when the spinal lesion is behind the third lumbar (L₃)
- Pain perception may be decreased or absent in the rear limbs; presence of deep pain sensation is the single most reliable prognostic

factor for return to acceptable function

- Forelimb function usually is normal
- Lack of ability to control urination (known as “urinary incontinence”) is common when the spinal lesion affects motor function
- Pain is less obvious in cats; the site of disk herniation often involves the lumbar vertebrae

CAUSES

- Degeneration of the thoracolumbar intervertebral disks
- 15% of animals with spinal fractures/dislocations have been reported to have disk extrusions, in addition to the fracture/dislocation

RISK FACTORS

- Hansen type I disease most often affects chondrodystrophic breeds (breeds with shortened legs that are bowed to some degree)



TREATMENT

HEALTH CARE

Guidelines for therapy based on classification of clinical condition, as follows:

- Class 1—back-pain only
- Class 2—back pain; wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”); mild weakness of the hind limbs (paraparesis); motor ability good
- Class 3—abnormalities in which normal subconscious awareness of the location of the limbs and movement is altered (known as “proprioceptive deficits”); motor ability affected, but still present
- Class 4—complete paralysis (no motor ability) of the hind limbs, with deep pain perception present
- Class 5—complete paralysis (no motor ability) of the hind limbs, no deep pain perception present

Treatment recommendations as follows:

- Class 1 patients treated medically, unless pain persists
- Class 2 patients treated medically initially with serial nervous system examinations, surgery if patient condition remains static or declines
- Classes 3 and 4 need surgical therapy
- Class 5 surgical therapy, if within the first 12 to 48 hours of occurrence
- Minimize spinal manipulation and support spine when handling patient
- Recumbent patients should be kept clean on padded bedding, placed on elevated cage racks, and turned frequently to prevent formation of “bed sores” (known as “decubital ulcers”)
- Ensure ability to urinate or consider bladder expression, intermittent catheterization, or indwelling urinary catheter for patients in classes 3 through 5
- Evacuation of the bowel or enemas may be necessary to promote defecation, as directed by your pet’s veterinarian
- Physical therapy with passive manipulation of rear limbs begun early followed by more intense therapy (hydrotherapy) for animals with nervous system deficits
- Carts are useful in many patients in promoting return to function; patient tolerance is limiting factor
- Acupuncture may be effective for animals with long-term (chronic) pain, where no compressive lesion can be identified by special contrast X-rays of the spine (known as “myelography”)
- Breaking up or dissolving the herniated intervertebral disk (known as “discolysis”) by enzymatic injection or laser ablation has been described, but is not a proven therapy in dogs

ACTIVITY

- Restricted activity and movement are most important part of medical management
- Cage rest in hospital or enforced cage rest as an outpatient for 2 to 4 weeks for class 1 patients or postoperative animals

DIET

- Weight reduction, if patient is overweight or obese

SURGERY

- Strongly indicated for animals in classes 3 and 4 and within the first 12 to 48 hours for class 5 dogs; also indicated for static or worsening class 1 and 2 dogs
- Primary surgical goal is to relieve spinal cord compression by removal of the herniated intervertebral disk (surgical procedures include

hemilaminectomy, dorsal laminectomy, and pediculectomy)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroid therapy may be beneficial; use of steroids is reserved for limited and specific cases
- Steroids given to animals without simultaneous strict cage confinement could worsen disk extrusion by encouraging exercise
- Nonsteroidal anti-inflammatory drugs (NSAIDs) or narcotics can be used to relieve pain (known as “analgesics”)
- Muscle relaxants (such as methocarbamol) may be useful in cases where muscle spasm is contributing to pain
- Bethanechol and phenoxybenzamine may be helpful in managing bladder dysfunction associated with spinal-cord lesions



FOLLOW-UP CARE

PATIENT MONITORING

- Serial nervous system examinations are important for all affected animals
- Patients treated medically should be re-evaluated 2 to 3 times daily for worsening nervous system signs for the first 48 hours after onset
- If stable, re-evaluate daily, then weekly, until clinical signs have resolved
- Patients treated surgically are evaluated frequently until improvement is noted; urinary bladder function is the limiting factor for hospitalization

PREVENTIONS AND AVOIDANCE

- Prevention of obesity and avoiding strenuous exercise or jumping may or may not avoid worsening of clinical signs

POSSIBLE COMPLICATIONS

- Recurrence of signs associated with disk herniation at original or new site of the spinal cord
- Deterioration of clinical signs with or without surgery; hard-to-predict clinical course in some cases, especially those with severe Hansen type I lesions
- Rarely, development of ascending or descending myelomalacia—condition in which the motor neurons (nerve cells that control muscles) are destroyed, leading to progressive spinal cord disease that is not reversible; occurs in class 4 or 5 dogs at 3 to 5 days following injury and characterized by variable and changing nervous system findings, possible fever, possible difficulty breathing (known as “dyspnea”); euthanasia should be performed when myelomalacia is diagnosed

EXPECTED COURSE AND PROGNOSIS

- Overall prognosis for dogs in classes 1 through 4 is good to excellent; those treated conservatively may experience recurrence of clinical signs
- Recurrence rates of dogs without fenestration at the time of surgical removal of the herniated disk material (laminectomy) range from 5% to 30%
- Dogs in class 5 have a variable chance of recovery; overall a guarded, but seemingly favorable prognosis if surgery is performed within 48 hours and the animal is allowed sufficient time to recover



KEY POINTS

- If clinical signs of nervous system disease become worse, the pet should be re-evaluated by the veterinarian as soon as possible
- Restricted activity and movement are most important part of medical management
- Cage rest in hospital or enforced cage rest as an outpatient for 2 to 4 weeks for class 1 patients or postoperative animals
- Most animals in classes 1 through 4 have a good to excellent prognosis for return to function (that is, ability to walk and have control of urination and bowel movements); patients in class 5 have a poorer, but not hopeless prognosis

INTUSSUSCEPTION



BASICS

OVERVIEW

- Folding of one segment of the intestine into another segment or invagination of one intestinal segment into the lumen of the adjacent segment
- Intussusceptions are classified according to their location within the gastrointestinal tract—"ileocolic" (involving the ileum, the last section of the small intestine and colon [large intestine]) and "jejunojejunal" (involving the jejunum, where both segments are in the jejunum, the middle section of the small intestine) intussusceptions are the types most commonly encountered in small animals; others that have been described include: "gastroesophageal" (involving the stomach and esophagus [tube from the throat to the stomach]); "duodenojejunal" (involving the duodenum [the upper or first section of the small intestine] and the jejunum [the middle section of the small intestine]), and "cecocolic" (involving the cecum [the blind pouch of the intestinal tract at the junction of the small intestine and the colon] and the colon)
- The segment entrapped within the lumen of the intussusception is called the "intussusceptum" and the engulfing segment is called the "intussusciens"
- Most commonly, the segment of the gastrointestinal tract closest to the mouth is found to be engulfed within the segment that is further away from the mouth; this typically means that the segment closest to the mouth is the intussusceptum and the segment further away from the mouth is the intussusciens

GENETICS

- German shepherd dogs appear to be more likely to have gastroesophageal intussusceptions than other breeds of dogs, accounting for approximately 60% of the reported cases of this condition
- German shepherd dogs also appear to be more likely than other dog breeds to have various types of intussusceptions
- Siamese may be more likely to have intussusceptions than other breeds of cats

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats, but more common in dogs

Breed Predilection

- German shepherd dogs appear to be more likely to have gastroesophageal intussusceptions than other breeds of dogs, accounting for approximately 60% of the reported cases of this condition
- German shepherd dogs also appear to be more likely than other dog breeds to have various types of intussusceptions
- Siamese may be more likely to have intussusceptions than other breeds of cats

Mean Age and Range

- Often younger animals due to risk factors (such as parasitism, viral inflammation of the intestines [known as "enteritis"], dietary indiscretion, foreign body ingestion) for development of an intussusception
- Older animals with intussusceptions should be screened carefully for diseases that cause alteration of the normal wave-like movement of the intestines (known as "peristalsis"), such as intestinal cancer

Predominant Sex

- Originally felt that males outnumbered females with gastroesophageal intussusception; recent reports have called this into question
- No documented sex predilection for other types of intussusceptions in small animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs associated with the intussusception depend on the anatomic location of the intussusception
- In general, intussusceptions occurring in sections of the gastrointestinal tract closer to the mouth have more severe clinical signs and disease progression; gastroesophageal intussusceptions typically cause more severe clinical signs than intussusceptions located in sections of the intestinal tract further away from the mouth
- Severity of clinical signs also depends upon the completeness of the blockage or obstruction; typically, complete blockage or obstruction of the gastrointestinal tract cause more severe clinical signs than partial blockage or obstruction
- Vomiting
- Diarrhea (which may or may not have fresh blood or black, tarry stools [known as "melena"] present)
- Abdominal pain
- Abdominal distention

- Lack of appetite (known as “anorexia”)
- Weight loss
- Signs may be sudden (acute) in onset or may have been occurring for weeks or months (chronic)
- May display overt abdominal pain/discomfort
- Depending on the severity of the intussusception, as well as the length of time that it has been present, some patients may be showing signs of heart and/or circulatory problems
- A sausage-shaped mass may be palpable in the abdomen; the ability to palpate the intussusception is variable
- Ileocolic intussusceptions may present with protrusion of gastrointestinal tissue from the rectum; this can be differentiated from a rectal prolapse via probing along the side of the protruding tissue—the presence of a blind-ending fold would indicate the existence of a rectal prolapse rather than an intussusception

CAUSES

- Any disease that alters gastrointestinal motility may lead to an intussusception
- Known causes include: inflammation of the intestines (enteritis); recent abdominal surgery; disease involving the wall of the intestinal tract (known as “intestinal mural disease”); and intestinal parasitism
- Intussusceptions occur in 8% to 33% of dogs that have kidney transplants; the reason is unclear, but may be related to immunosuppressive drugs

RISK FACTORS

- Disease processes that alter intestinal motility



TREATMENT

HEALTH CARE

- Initial efforts should be focused on patient stabilization, as well as correction of dehydration and existing electrolyte abnormalities
- Intravenous fluid administration to correct dehydration as well as to replace anticipated ongoing losses through vomiting and diarrhea
- Typically isotonic crystalloids are used; specific choice of fluid type is dictated by specific electrolyte abnormalities identified on blood work, as well as the veterinarian’s preference; crystalloids are fluids that contain electrolytes (chemical compounds, such as sodium, potassium, chloride) necessary for the body to function, crystalloids generally are similar to the fluid content (plasma) of the blood and move easily between the blood and body tissues, example is lactated Ringer’s solution

ACTIVITY

- Controlled activity (leash walks only, no running/jumping) for 10-to-14 days postoperatively, to prevent some complications

DIET

- If patient is actively vomiting—no food or water by mouth
- If vomiting is occurring postoperatively, ileus (a type of intestinal obstruction, caused by lack of normal intestinal motility) may be an underlying cause
- Many patients can be fed within 24 hours of surgical correction

SURGERY

- Surgical correction should be performed as soon as the patient is stable enough to withstand anesthesia and surgery—intussusception is a surgical emergency
- A full abdominal exploratory should be performed to assist in the identification of any potential underlying causes; also, multiple intussusceptions may be present in one patient at the same time
- Some intussusceptions can be reduced manually by gentle manipulation during surgical exploration of the abdomen; upon reduction, the bowel may or may not be able to live (known as being “viable”)
- In the event that manual reduction is not possible, or the bowel has questionable viability, a surgical procedure (known as an “intestinal resection and anastomosis” in which the diseased portion of the intestinal tract is removed surgically and the ends are sutured together) is necessary
- Enteroplication (a surgical procedure in which the intestines are folded or “pleated”) has been proposed to attempt to prevent another intussusception



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Use of antibiotics is recommended; the choice of antibiotics used should be dictated by the bacteria that are anticipated to be encountered
- Antibiotics are not recommended greater than 24 hours postoperatively, except in cases in which infection and inflammation of the lining of the abdomen (known as “septic peritonitis”) is present either preoperatively or postoperatively
- Postoperatively, patients should be maintained on intravenous fluids and pain medications should be administered



FOLLOW-UP CARE

PATIENT MONITORING

- Most recurrences occur with the first few days of surgery, but recurrences have been reported up to 3 weeks after surgery
- Intestinal dehiscence is a condition in which the surgery site splits or bursts open; it typically occurs 3-to-5 days postoperatively; infection and inflammation of the lining of the abdomen (septic peritonitis) will result and these patients have to be identified and addressed immediately

PREVENTIONS AND AVOIDANCE

- Prevention of many of the underlying causes can be achieved through such actions as vaccination against parvovirus, intestinal parasite control, limiting situations where patients can be exposed to dietary indiscretion or foreign body ingestion

POSSIBLE COMPLICATIONS

- Recurrence—6% to 27% of patients
- Infection and inflammation of the lining of the abdomen (septic peritonitis)—may result from postoperative intestinal dehiscence (condition in which the surgery site splits or bursts open) or bacterial contamination of the abdomen prior to or during surgery
- “Short-bowel syndrome” is a rare complication that can occur when significant intestinal length is removed surgically (generally more than 70% of the intestine removed in dogs)

EXPECTED COURSE AND PROGNOSIS

- Highly dependent upon underlying cause, location of intussusception, condition at presentation, response to therapy
- Generally, as the location of the intussusception is further away from the mouth, the prognosis improves as these patients are not as affected clinically
- Gastroesophageal intussusceptions have a grave prognosis, with mortality rates approaching 95%
- Intestinal intussusceptions generally have a good prognosis



KEY POINTS

- Immediate surgical intervention is the recommended treatment for intussusceptions—they are considered surgical emergencies
- Intussusceptions can occur in dogs and cats secondary to a wide variety of underlying conditions that alter intestinal motility; identify and treat the underlying cause—in young patients, it is typically inflammation of the intestines (enteritis) or intestinal parasites; in older patients, it is caused more commonly by either cancer or inflammation in the walls of the intestines
- Complications may include death; infection and inflammation of the lining of the abdomen (septic peritonitis); protracted hospital stay to stabilize; and recurrence
- Recurrence rates have been reported to be between 6% and 27%

IRRITABLE BOWEL SYNDROME (“IBS”)



BASICS

OVERVIEW

- A condition characterized by long-term (chronic) intermittent signs of abnormal function of the large intestines (colon), in the absence of structural gastrointestinal disease
- “Gastro-” refers to stomach; “intestinal” refers to the intestines
- “Bowel” refers to the intestines

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Any breed; especially working dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Long-term (chronic), intermittent signs of large-bowel diarrhea, including frequent passage of small amounts of bowel movement (feces) and mucus, and difficulty defecating (known as “dyschezia”)
- Passage of blood in the bowel movement or stool (known as “hematochezia”) is uncommon.
- Abdominal pain, bloating, vomiting, and nausea may occur
- Physical examination is often unremarkable
- Rectal examination is normal, aside from large-bowel diarrhea

CAUSES

- Unknown

RISK FACTORS

- Stress (such as changes in the household or being left alone for extended periods) may be associated with episodes of diarrhea
- In many dogs, stress appears to play no role



TREATMENT

HEALTH CARE

- Outpatient medical management

DIET

- A highly digestible diet with added soluble fiber often improves diarrhea, but rarely completely resolves clinical signs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drug therapy for several days up to 1 to 2 weeks during episodes
- Sulfasalazine (Azulfidine®)—medication that has antibacterial and anti-inflammatory properties; reported to improve signs in some dogs with significant difficulty defecating (dyschezia)

Intestinal Motility Modifiers

- Opiate antidiarrheals improve signs by increasing rhythmic segmentation in the intestines
- Loperamide (Imodium®)
- Diphenoxylate (Lomotil®)

Antispasmodic–Tranquilizer Combinations

- Used to relieve abdominal cramping, bloating, and distress
- Chlordiazepoxide and clidinium bromide (Librax®)
- Isopropamide and prochlorperazine (Darbazine®)

Medications to Control Nausea and Vomiting Administered by Injection (Known as “Parenteral Antiemetics”)

- If nausea and vomiting preclude administering medication by mouth, administer antiemetics by injection for 1 to 2 days
- Chlorpromazine (Thorazine®)



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor stool consistency and watch for signs of difficulty defecating (dyschezia) and abdominal discomfort—call your pet’s veterinarian if you observe changes in stool consistency or any signs of large-bowel problems

PREVENTIONS AND AVOIDANCE

- Minimize any stressful factors in the pet’s environment that might precipitate an episode

EXPECTED COURSE AND PROGNOSIS

- Should see improved stools, decreased mucus, and relief of difficulty defecating (dyschezia) and abdominal distress within 1 to 2 days of starting medication
- In some dogs, signs completely resolve following treatment and dietary alterations; others have long-term episodic signs



KEY POINTS

- Response to treatment varies and affected dogs may have long-term, intermittent clinical signs
- Eliminate any stressful factors in the dog’s environment if possible

JOINT LUXATIONS (DISLOCATED JOINTS)



BASICS

OVERVIEW

- “Luxation” is the medical term for dislocation; it refers to the complete disruption of a joint when the supporting structures (such as ligaments) around the joint are damaged or missing; “subluxation” refers to a partial dislocation or disruption of a joint
- “Laxity” describes the degree of abnormal looseness in the motion of a joint; the greater the looseness, the greater is the likelihood of joint injury, including sprains and partial or complete dislocations

GENETICS

- “Hyperlaxity syndrome” is an inherited disorder in people, in which multiple joints are very loose; puppies may show temporary extreme looseness (hyperlaxity) of the joints, when confined for long periods of time
- Hip dysplasia is a form of inherited looseness (laxity) of the hip joint; development of hip dysplasia is determined by interaction of genetic and environmental factors
- Shoulder dislocation or luxation is an inherited susceptibility in small breed dogs, like the miniature poodle
- “Ehlers-Danlos syndrome” is a group of inherited connective-tissue disorders, in which the skin is very elastic and the joints are highly moveable and loose

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Varies with the joint affected
- Hip—large-breed dogs show clinical signs of hip dysplasia more frequently than smaller breed dogs, but breeds of all sizes can have bone changes characteristic of hip dysplasia on X-rays; cats are affected by hip dysplasia, but it is much less common than in dogs
- Congenital (present at birth) shoulder dislocation (luxation) is more common in miniature breeds, such as the poodle

Mean Age and Range

- Traumatic—any age
- Congenital (present at birth) joint laxity may be seen at 4 to 12 months, with related inflammation of the joint (known as “arthritis” or “degenerative joint disease”) showing up later in life

SIGNS/OBSERVED CHANGES in the ANIMAL

- Varies with the joint affected
- Abnormal anatomic or structural position of one bone in relation to the adjoining bone
- Swelling, pain and non-use of the limb usually are seen initially; partial weight bearing may occur with time
- Traumatic dislocations (luxations) may occur at any joint
- Spinal dislocations (luxations) occur as a result of trauma, with sometimes devastating results (such as complete paralysis)
- Stifle or knee—cranial cruciate ligament rupture (known as a “ruptured cruciate”) leads to instability and partial dislocation (subluxation) of the stifle; animal may limp or may carry the affected rear leg

CAUSES

- Trauma—displacement of normal joint tissues beyond their elastic limit
- Minimal stress applied to abnormally unstable joints in dogs having joint problems with congenital (present at birth) causes

RISK FACTORS

- Abnormal conformation, causing increased stresses on the joint
- Fatigue, causing muscle weakness and incoordination
- Nervous-system abnormalities



TREATMENT

HEALTH CARE

- Rest, reduce mobility, reduce swelling, control pain
- Stabilize the joint (may be able to restore the joint alignment under anesthesia [“closed reduction”] or may require surgical correction [“open reduction”] under anesthesia) or salvage the limb by removing the source of pain
- Bandage, if the affected joint is at the elbow or lower in the foreleg or at the stifle (knee) or lower in the rear leg
- Cold compresses for 5 to 10 minutes, 4 or 5 times a day initially

ACTIVITY

- Cage rest, until joint stabilization

DIET

- Normal

SURGERY

- Closed reduction under anesthesia may be successful if the support structures are intact and no anatomic aberrations are present; “closed reduction” is the restoration of the joint alignment without surgically entering the joint—the veterinarian will manipulate the bones in such a manner as to return them to their normal positions within the joint
- Failing closed reduction, an “open” surgical approach or “open reduction” may be used; in this case, the veterinarian manipulates the bones while observing the bones and the joint during surgery; the joint is reduced or restored—after reduction, some form of surgical stabilization should be applied to reduce the possibility of the dislocation recurring; after surgical closure, an external support sling often is used to limit movement until the tissues around the joint have healed
- The incidence of the dislocation recurring (reluxation) is high, especially in the case of dislocations (luxations) of congenital (present at birth) cause
- Salvage procedures include prosthetic joint replacement (such as a total hip replacement) where available; surgical removal of bone-to-bone contact points (such as surgical removal of the “ball” of the “ball and socket” hip joint [known as a “femoral head and neck ostectomy”]); and fusing the joint (known as “arthrodesis”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs) to decrease pain and inflammation in the joint
- Analgesics to decrease pain



FOLLOW-UP CARE

PATIENT MONITORING

- Always take an X-ray after restoration (reduction) of the joint
- Take follow-up X-rays when the sling is removed, and a few weeks later

PREVENTIONS AND AVOIDANCE

- Keep pet in fenced-in yards
- Keep the sling in place until healing has occurred

POSSIBLE COMPLICATIONS

- Recurrence of the dislocation (luxation) or partial dislocation (subluxation)
- Infection after surgery
- Failure of prosthetic device for joint replacement
- Inflammation of the joint (arthritis)

EXPECTED COURSE AND PROGNOSIS

- Return of function is expected, unless a complication occurs
- High incidence of recurrence of the dislocation (luxation) or partial dislocation (subluxation) makes the prognosis guarded



KEY POINTS

- Abnormal anatomic or structural position of one bone in relation to the adjoining bone
- Swelling, pain and non-use of the limb usually are seen initially; partial weight bearing may occur with time
- High incidence of recurrence of the dislocation (luxation) or partial dislocation (subluxation) makes the prognosis guarded
- Activity and body weight gains increase the likelihood of developing degenerative joint changes (arthritis) in the long term

NONULCERATIVE KERATITIS

(TYPE OF INFLAMMATION OF THE CORNEA)



BASICS

OVERVIEW

- “Keratitis” is inflammation of the cornea; the “cornea” is the clear outer layer of the front of the eye
- “Nonulcerative keratitis” is any inflammation of the cornea that does not retain fluorescein stain; “fluorescein stain” is a dye that is used to identify ulcers of the cornea—if the very top layer of the cornea has been disrupted (as with an ulcer), the dye will enter the lower layers of the cornea and will cause a temporary stain that glows under an ultraviolet light; in nonulcerative keratitis, the top layer of the cornea is not disrupted, so no dye enters the lower layers of the cornea

GENETICS

- No proven genetic basis in dogs or cats
- Long-term (chronic) superficial inflammation of the cornea (keratitis), also known as “pannus”—inherited susceptibility considered in the German shepherd dog

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Long-term (chronic) superficial inflammation of the cornea (keratitis), also known as “pannus”—may occur in any breed; high likelihood in German shepherd dogs and Belgian Tervuren
- Inflammation characterized by the presence of pigment (melanin) that is deposited in the cornea (known as “pigmentary keratitis”)—seen in short-nosed, flat-faced (known as “brachycephalic”) breeds of dogs with inflammation of the cornea due to exposure to air and irritants (known as “exposure keratopathy”) from condition in which the eyelids do not close completely (known as “lagophthalmos”) and tear-film deficiencies; prominent folds of skin around the nose; abnormal eyelashes that turn inward, against the cornea (known as “trichiasis”); notably identified in pugs, Lhasa apsos, shih tzus, Pekingese
- Inflammation usually involving the area where the cornea (clear part of the eye) and the sclera (white part of the eye) come together, characterized by the presence of nodules (condition known as “nodular granulomatous episcleritis”)—may occur in any breed; likely in cocker spaniels, greyhounds, collies and Shetland sheepdogs
- “Dry eye” (known as “keratoconjunctivitis sicca” or “KCS”)—seen in short-nosed, flat-faced (brachycephalic) breeds; notably cocker spaniels, English bulldogs, Lhasa apsos, shih tzus, pugs, West Highland white terriers, Pekingese, Cavalier King Charles spaniels
- Condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up (known as edema; condition known as “corneal sequestration”)—most prevalent in Persians, Siamese, Burmese, and Himalayans

Mean Age and Range

- Long-term (chronic) superficial inflammation of the cornea (keratitis), also known as “pannus”—may occur at any age; higher risk at 4 to 7 years of age
- Inflammation characterized by the presence of pigment (melanin) that is deposited in the cornea (pigmentary keratitis)—may occur at any age
- Inflammation usually involving the area where the cornea (clear part of the eye) and the sclera (white part of the eye) come together, characterized by the presence of nodules (nodular granulomatous episcleritis)—may occur at any age; in collies—young to middle-aged (mean age, 3.8 years)
- “Dry eye” (keratoconjunctivitis sicca or KCS)—usually middle-aged or old
- Herpesvirus in cats—all ages
- Inflammation of the cornea, characterized by the presence of a type of white-blood cell, called an “eosinophil” (condition known as “eosinophilic keratitis”) and condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up (known as edema; condition known as “corneal sequestration”) in cats—all ages, except newborns

SIGNS/OBSERVED CHANGES in the ANIMAL

- May cause variable discoloration of the cornea
- Variable eye discomfort

Dogs

- Long-term (chronic) superficial inflammation of the cornea (keratitis), also known as “pannus”—usually involves both eyes; often symmetrical pinkish white lesions with variable pigmentation; usually seen on the outer and/or lower part of the cornea; third eyelids

may be affected and appear thickened or depigmented; white lipid (a group of compounds that contain fats or oils) deposits may be present at adjacent corneal edge; may lead to blindness in advanced disease

- Inflammation characterized by the presence of pigment (melanin) that is deposited in the cornea (pigmentary keratitis)—appears as focal to diffuse brown to black discoloration of the cornea; often associated with encroachment of blood vessels into corneal tissue (known as “corneal vascularization”) or scarring
- Inflammation usually involving the area where the cornea (clear part of the eye) and the sclera (white part of the eye) come together, characterized by the presence of nodules (nodular granulomatous episcleritis)—usually involves both eyes; raised pink to tan lesions of the outer part of the cornea; may be slowly to rapidly progressive; white deposits and encroachment of blood vessels into corneal tissue (corneal vascularization) may occur in adjacent corneal tissue; third eyelids may appear thickened
- “Dry eye” (keratoconjunctivitis sicca or KCS)—variable findings; may involve one or both eyes; discharge from the eye(s) may contain mucus and/or pus; redness of the moist tissues of the eye (known as “conjunctival hyperemia”); encroachment of blood vessels into corneal tissue (corneal vascularization); pigmentation; and variable scarring

Cats

- Herpesvirus (nonulcerative; involves the thick, clear middle layer of the cornea [known as the “stroma”])—may involve one or both eyes; often occurs with ulceration; fluid build-up in the cornea (known as “corneal edema”), infiltrates, encroachment of blood vessels into corneal tissue (corneal vascularization), scarring; may threaten vision, if severe scarring
- Inflammation of the cornea, characterized by the presence of a type of white-blood cell, called an “eosinophil” (condition known as “eosinophilic keratitis”)—usually involves only one eye; appears as raised white, pink, or gray corneal plaque with roughened surface; may retain fluorescein stain at the edge of the lesion
- Condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up (known as edema; condition known as “corneal sequestration”)—usually involves only one eye, but can involve both eyes; appears as amber, brown, or black oval to circular plaques near the center of the cornea; can vary in size and corneal depth; edges may appear raised because of fluid build-up in the cornea (corneal edema); thickened tissue; encroachment of blood vessels into corneal tissue (corneal vascularization) is variable; may retain fluorescein at edge of lesion

CAUSES

Dogs

- Long-term (chronic) superficial inflammation of the cornea (keratitis), also known as “pannus”—presumed to be immune-mediated; altitude and solar radiation increase the likelihood and severity of the disease
- Inflammation characterized by the presence of pigment (melanin) that is deposited in the cornea (pigmentary keratitis)—secondary to any long-term (chronic) corneal irritation; evaluate for primary underlying eye conditions; more frequently associated with exposure corneal disease (exposure keratopathy) and “dry eye” (keratoconjunctivitis sicca or KCS)
- Inflammation usually involving the area where the cornea (clear part of the eye) and the sclera (white part of the eye) come together, characterized by the presence of nodules (nodular granulomatous episcleritis)—presumed to be immune-mediated
- “Dry eye” (keratoconjunctivitis sicca or KCS)—usually caused by immune-mediated inflammation of the lacrimal gland that produces tears (condition known as “dacryoadenitis”)

Cats

- Herpesvirus—believed to be immune-mediated reaction to herpesvirus antigen rather than an actual effect of the viral infection
- Inflammation of the cornea, characterized by the presence of a type of white-blood cell, called an “eosinophil” (condition known as “eosinophilic keratitis”)—unknown; some cats are infected with herpesvirus
- Condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up (known as edema; condition known as “corneal sequestration”)—unknown; likely due to long-term (chronic) corneal irritation; some cats have history of previous trauma; suggested relationship with previous herpesvirus infection

RISK FACTORS

- Dogs—long-term (chronic) superficial inflammation of the cornea (keratitis), also known as “pannus”—more likely to occur at high altitudes with intense sunlight



TREATMENT

HEALTH CARE

- Outpatient—generally sufficient
- Inpatient—cases that warrant surgery due to inadequate response to medical therapy
- Long-term (chronic) superficial inflammation of the cornea (keratitis), also known as “pannus”—radiation therapy (using β -irradiation with a strontium-90 probe)
- Inflammation characterized by the presence of pigment (melanin) that is deposited in the cornea (pigmentary keratitis)—radiation therapy (using β -irradiation) and freezing (known as “cryotherapy”)

SURGERY

Dogs

- Long-term (chronic) superficial inflammation of the cornea (keratitis), also known as “pannus”—surgical removal of the surface of the cornea (known as “superficial keratectomy”) may be performed for severe disease; usually unnecessary; even if surgery is performed, still requires indefinite medical treatment to prevent recurrence
- Inflammation characterized by the presence of pigment (melanin) that is deposited in the cornea (pigmentary keratitis)—surgical removal of the surface of the cornea (superficial keratectomy) may be performed only after initial underlying cause is corrected; surgery only in severe cases in which inflammation threaten vision
- Inflammation usually involving the area where the cornea (clear part of the eye) and the sclera (white part of the eye) come together, characterized by the presence of nodules (nodular granulomatous episcleritis)—surgical removal of the surface of the cornea (superficial keratectomy) is diagnostic; usually unnecessary; only temporarily resolves clinical signs; medical treatment still is required
- “Dry eye” (keratoconjunctivitis sicca or KCS)—surgically move the duct from the parotid salivary gland to the eye (procedure known as a “parotid duct transposition”), the saliva then acts as “tears” in the eye, or permanent partial closure of the eyelids (surgical procedure known as a “tarsorrhaphy”) may be indicated

Cats

- Inflammation of the cornea, characterized by the presence of a type of white-blood cell, called an “eosinophil” (condition known as “eosinophilic keratitis”)—surgical removal of the surface of the cornea (superficial keratectomy) is diagnostic; usually unnecessary; only temporarily resolves clinical signs; medical treatment is preferred
- Condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up (known as edema; condition known as “corneal sequestration”)—surgical removal of the surface of the cornea (superficial keratectomy) may be curative; recurrence is possible; eye discomfort is primary indication for surgery



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Dogs

- Long-term (chronic) superficial inflammation of the cornea (keratitis), also known as “pannus”—steroids (1% prednisolone or 0.1% dexamethasone) applied to the eye directly (known as “topical treatment”); 1% or 2% cyclosporine in oil or 0.2% ointment to decrease the immune response, applied to the eye directly (topical treatment); either of these medications can be used alone or in combination for more severe cases; steroid (triamcinolone) injection under the moist tissues of the eye (known as “subconjunctival injection”) can be used in addition to topical therapy in severe cases
- Inflammation characterized by the presence of pigment (melanin) that is deposited in the cornea (pigmentary keratitis)—medications applied to the eye directly (topical treatment) to treat underlying cause; topical steroids, if primary cause is inflammatory; lubricants or cyclosporine, if primary condition is “dry eye” (keratoconjunctivitis sicca or KCS); cyclosporine may be beneficial to reduce pigmentation
- Inflammation usually involving the area where the cornea (clear part of the eye) and the sclera (white part of the eye) come together, characterized by the presence of nodules (nodular granulomatous episcleritis)—steroids and/or cyclosporine applied to the eye directly (topical treatment); systemic azathioprine (a chemotherapeutic agent used to decrease the immune response) may be effective when used alone or in combination with topical medications
- “Dry eye” (keratoconjunctivitis sicca or KCS)—topical 1% or 2% cyclosporine in oil or 0.2% ointment

Cats

- Herpesvirus—topical (applied to the eye directly) antiviral agents (such as trifluridine—Viroptic®); for disease of the thick, clear middle layer of the cornea (the stroma); topical steroids can be used at the same time as antiviral agents, but with caution; oral lysine may be of benefit; oral antiviral agents should be used with extreme caution because of bone-marrow suppression, leading to low red-blood cell and low white-blood cell counts, that could proceed to death
- Inflammation of the cornea, characterized by the presence of a type of white-blood cell, called an “eosinophil” (condition known as “eosinophilic keratitis”)—steroids (1/8 to 1% prednisolone or 0.1% dexamethasone) applied to the eye directly (topical treatment) usually causes remission; steroids should be used with caution and the patient monitored for ulceration or worsening of clinical signs; topical antiviral medications can be used in combination with steroids, if herpesvirus infection is suspected; for severe cases that do not respond to medical treatment, megestrol acetate (Ovaban®) can be considered; megestrol acetate has side effects that you should discuss with your pet’s veterinarian
- Condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up (known as edema; condition known as “corneal sequestration”)—triple antibiotic applied to the eye directly (topical treatment) as directed by your pet’s veterinarian for associated corneal ulceration; artificial tear lubrication may be beneficial for relieving discomfort; topical antiviral medications can be used, if herpesvirus infection is suspected; topical 1% atropine ointment may be used to treat pain associated with coexistent inflammation of the front part of the eye, including the iris (known as “anterior uveitis”), if clinical signs suggestive of uveitis are

present



FOLLOW-UP CARE

PATIENT MONITORING

- Periodic eye examinations to evaluate effectiveness of treatment; examine at 1 to 2 week intervals, gradually lengthening the interval with remission or resolution of clinical signs

POSSIBLE COMPLICATIONS

- Continued eye discomfort
- Visual defects
- Blindness in severe cases

EXPECTED COURSE AND PROGNOSIS

- Depend on disease and underlying cause



KEY POINTS

Dogs

- All patients require lifelong treatment
- Nonulcerative keratitis is controlled rather than cured
- Surgery may be needed for treatment; some animals will continue to need medical treatment following surgery

Cats

- Herpesvirus—eye discomfort and inflammation of the cornea (keratitis) often recur
- Inflammation of the cornea, characterized by the presence of a type of white-blood cell, called an “eosinophil” (condition known as “eosinophilic keratitis”)—disease controlled rather than cured
- Condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up (known as edema; condition known as “corneal sequestration”)—the pigmented lesion (known as a “sequestrum”) may slough spontaneously; may require months to years of treatment and clinical course may be prolonged without surgery; removal of sequestrum by surgical removal of the surface of the cornea (superficial keratectomy) may be incomplete and it may recur postoperatively

ULCERATIVE KERATITIS

(TYPE OF INFLAMMATION OF THE CORNEA)



BASICS

OVERVIEW

- “Keratitis” is inflammation of the cornea; the “cornea” is the clear outer layer of the front of the eye
- The “corneal epithelium” is the top surface layer of the cornea; the “corneal stroma” is the thick, clear middle layer of the cornea; the “corneal endothelium” is the inner lining layer of the cornea
- “Ulcerative keratitis” is inflammation of the cornea associated with loss of the top surface of the cornea (corneal epithelium; condition known as a “corneal erosion”) and possibly loss of variable amounts of the underlying thick, clear middle layer of the cornea (known as the “stroma;” condition known as a “corneal ulcer”)
- Ulcerative keratitis is identified by retention of fluorescein stain in the corneal erosions or ulcers; “fluorescein stain” is a dye that is used to identify ulcers of the cornea—if the very top layer of the cornea has been disrupted (as with an erosion or ulcer), the dye will enter the lower layers of the cornea and will cause a temporary stain that glows under an ultraviolet light

GENETICS

- No proven basis, although certain breeds appear to be more likely to have ulcerative keratitis
- May occur secondary to other corneal diseases that have breed predispositions and presumably a genetic basis, such as degeneration of the top surface of the cornea, leading to erosions or ulcers (condition known as “corneal epithelial dystrophy”) in Shetland sheepdogs and degeneration of the lining of the cornea, leading to progressive fluid build-up in the cornea (known as “corneal edema;” condition known as “corneal endothelial dystrophy”) in Boston terriers

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dogs—short-nosed, flat-faced (known as “brachycephalic”) breeds are more likely than other breeds to develop ulcerative keratitis
- Spontaneous, long-term defects in the top surface of the cornea (known as “chronic corneal epithelial defects”)—occurs in any breed
- Cats—Persian, Himalayans, Siamese, and Burmese are more likely to develop a condition in which part of the cornea tissue dies, leaving a pigmented lesion and fluid build-up (known as “edema;” condition known as “feline corneal sequestration”) than other cat breeds

Mean Age and Range

- Age of onset—variable; determined by cause
- Spontaneous, long-term defects in the top surface of the cornea (chronic corneal epithelial defects)—middle-aged and older dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- May be sudden (acute) or long-term (chronic)
- Tearing, squinting, rubbing at eyes
- Appearance of a “film over the eye” (often due to fluid build-up in the tissues of the cornea [known as “corneal edema”]); prolapsed third eyelid
- Sometimes history of trauma
- Herpetic ulcers (cats)—may have history of upper respiratory infection (URI)
- Discharge from the eye(s); may be clear or may contain mucus and/or pus
- Squinting or spasmodic blinking (known as “blepharospasm”)
- Avoidance of light (known as “photophobia”)
- Redness of the moist tissues of the eye (known as “conjunctival hyperemia”)
- Superficial inflammation of the cornea (keratitis)—may note one or more circumscribed, linear, or geographic defects in the cornea
- Deep stromal ulcer (involving the thick, clear middle layer of the cornea [known as the “stroma”]) or extremely deep ulceration of the cornea, to the inner most membrane of the cornea (known as a “descemetocoele”)—may appear as a “crater-like” defect
- Depending on cause and duration—may see encroachment of blood vessels into corneal tissue (known as “corneal vascularization”); pigmentation; scarring; mineral or lipid (a group of compounds that contain fats or oils) deposition in the corneal tissue; inflammatory cell infiltrate (yellow to cream-colored opacity with indistinct margins, often surrounded by fluid build-up [corneal edema]) in the

corneal tissue; collagenolytic activity (melting) of the thick, clear middle layer of the cornea (the stroma)

- Spontaneous, long-term defects in the top surface of the cornea (chronic corneal epithelial defects)—loose or redundant epithelial edges; may see fluorescein stain extending into areas with seemingly intact epithelium (ring of less intense staining)
- Ulcerative corneal disease usually stimulates tear production; absence of obvious tearing suggests pet may have “dry eye” (known as “keratoconjunctivitis sicca” or “KCS”)
- Inflammation of the front part of the eye, including the iris (known as “anterior uveitis”)—mild or severe, secondary to ulceration; severe anterior uveitis may result in accumulation of white-blood cells in the anterior chamber of the eye (condition known as “hypopyon”); severe anterior uveitis suggests coexistent bacterial infection

CAUSES

- Trauma—blunt; penetrating; perforating
- Disease of tissues surrounding the eye (known as “adnexa”), such as eyelids, third eyelid, and tear glands
- Inability to close eyelids completely (known as “lagophthalmos”)—results in inflammation of the cornea due to exposure to air and irritants (known as “exposure keratitis”); may be breed-related in short-nosed, flat-faced (brachycephalic) dogs and cats
- Tear-film abnormality—quantitative tear deficiency (such as in “dry eye” [keratoconjunctivitis sicca or KCS]); qualitative tear film deficiency caused by mucin deficiency or some other unidentified tear abnormality
- Infection—usually secondary in dogs; can be primary in cats (herpesvirus infection)
- Primary corneal disease—degeneration of the lining of the cornea (endothelial dystrophy); other diseases of the lining of the cornea
- Miscellaneous—foreign body (corneal or conjunctival); chemical burns; loss of nerve (trigeminal nerve) sensation; immune-mediated disease



TREATMENT

HEALTH CARE

- Hospitalize patients with deep or rapidly progressive ulcers; these may require surgery and/or frequent medical treatments
- Keep facial hair out of eyes

ACTIVITY

- Restrict in patients with deep stromal ulcers (involving the thick, clear middle layer of the cornea [the stroma]) or extremely deep ulceration of the cornea, to the inner most membrane of the cornea (known as “Descemet’s membrane,” condition known as a “descemetocoele”) to prevent rupture of the cornea (eye)
- Prevent self-trauma with Elizabethan collar

SURGERY

- Superficial ulcers do not usually require surgery, if the underlying cause has been eliminated
- Ulcer that extends one-half or greater of the corneal thickness and particularly to the inner most membrane of the cornea (known as “Descemet’s membrane,” condition known as a “descemetocoele”) may benefit from surgery
- Extremely deep ulceration of the cornea, to the inner most membrane of the cornea (descemetocoele) or full thickness corneal laceration—considered a surgical emergency

Procedures

- Spontaneous long-term (chronic) corneal epithelial defects—removal of loose epithelium after application of anesthesia directly to the eye (known as “topical anesthesia”); punctate or grid incisions into the cornea (procedure known as a “keratotomy”) are performed easily after removal of loose epithelium with topical anesthesia; surgical removal of the surface of the cornea (known as “superficial keratectomy”) is more invasive and may cause more scarring; application of a contact lens or third-eyelid flap after any of these procedures will improve comfort and aid healing
- Therapeutic contact lens placement—acts as a bandage to reduce both frictional irritation from the eyelids and pain; most useful in cases with spontaneous long-term (chronic) corneal epithelial defects; easy application and can still monitor and examine the eye; most retained 1 to 2 weeks, then removed; should monitor eye for increased pain and fluid build-up in the cornea (corneal edema), which may indicate that the contact lens does not fit and is causing corneal injury
- Various surgical procedures (such as rotational pedicle conjunctival flap, corneoscleral transposition, corneal transplant) may be performed for cases with ulcers greater than 50% thickness of the thick, clear middle layer of the cornea (the stroma) and extremely deep ulceration of the cornea, to the inner most membrane of the cornea (descemetocoele)
- Cyanoacrylate repair (corneal glue)—can be used for deep ulcers; promotes encroachment of blood vessels into corneal tissue (corneal vascularization) and stabilizes the cornea, but has somewhat lower success rate compared to corneal surgery



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antibiotics

- Antibiotics applied to the eye directly (known as “topical antibiotics”)—indicated for all patients
- Frequency of application—determined by severity of the corneal disease and the type (ointment, liquid solution) of topical antibiotic used; ointments have a relatively long contact time; solutions are applied more frequently
- Commonly used topical antibiotics—erythromycin (cats); triple antibiotic, gentamicin, and tobramycin
- Uncomplicated ulcers or superficial erosions—combination of neomycin, polymyxin B, and bacitracin (that is, triple antibiotic) is an excellent first choice; broad spectrum of antimicrobial activity
- Complicated ulcers—often use combination therapy of cefazolin with either an aminoglycoside (tobramycin, gentamicin) or fluoroquinolone (ciprofloxacin, ofloxacin); particularly in rapidly progressive, deep, or melting ulcers; frequency depends on severity

Atropine

- 1% ointment or solution applied to the eye directly (topical treatment)
- Indicated for inflammation of the front part of the eye, including the iris (anterior uveitis)

Antiviral Agents

- Indicated for herpetic ulcers in cats
- Trifluridine (Viroptic®) solution

Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

- May be indicated for anti-inflammatory and pain-relieving (known as “analgesic”) properties

Other Medications

- Acetylcysteine—anti-collagenolytic agent used for treatment of melting ulcers
- Autologous serum—anti-collagenolytic agent; keep refrigerated; avoid contamination; discard after 48 hours



FOLLOW-UP CARE

PATIENT MONITORING

- Superficial ulcers—repeat fluorescein stain in 3 to 6 days; if ulcer persists 7 days or longer, either inciting cause has not been eliminated or the patient has spontaneous long-term (chronic) corneal epithelial defects
- Deep stromal (involving the thick, clear middle layer of the cornea [the stroma]) or rapidly progressive ulcers—assess every 24 hours initially if outpatient until improvement is seen; many of these patients are hospitalized or undergo surgery

PREVENTIONS AND AVOIDANCE

- Short-nosed, flat-faced (brachycephalic) dogs—lubricant ointment administration, permanent partial closure of the eyelids (surgical procedure known as a “tarsorrhaphy”) or both may help prevent recurrent ulceration
- “Dry eye” (keratoconjunctivitis sicca or KCS)-related ulcers—life-long treatment of KCS with cyclosporine or surgical movement of the duct from the parotid salivary gland to the eye (procedure known as a “parotid duct transposition”), the saliva then acts as “tears” in the eye, to prevent continued ulceration
- Herpesvirus (cats)—may try oral lysine to prevent viral replication; may decrease severity and/or frequency of outbreaks

POSSIBLE COMPLICATIONS

- Progressive corneal ulceration—rupture of eyeball
- Bacterial infection/inflammation of the tissues within the eyeball (known as “endophthalmitis”)
- Secondary glaucoma (in which the pressure within the eye [intraocular pressure] is increased secondary to inflammation in the front part of the eye)
- Softening and loss of tissue of the eyeball (known as “phthisis bulbi”)
- Blindness
- Blind and painful eye (may require surgical removal of the eyeball [known as “enucleation”])

EXPECTED COURSE AND PROGNOSIS

- Uncomplicated superficial ulcer—usually heals in 5 to 7 days
- Spontaneous long-term (chronic) corneal epithelial defects—may persist for weeks to months; may require multiple treatment procedures
- Deep corneal ulcer treated medically—may require several weeks for repair of defect; does not always heal satisfactorily; continued deterioration of ulcer and eyeball (globe) rupture are possible
- Deep ulcer treated with conjunctival flap—frequently results in more comfort within a few days after surgery



KEY POINTS

- Wait at least 5 minutes between medications, if more than one eye medication is applied to the eye directly (topical treatment); wait longer between ointments
- Contact your pet's veterinarian if patient appears more painful or the eye markedly changes in appearance
- Spontaneous long-term (chronic) corneal epithelial defect—discuss prolonged course of disease with your pet's veterinarian; usually achieve healing within 2 to 6 weeks, but may require weekly rechecks and multiple procedures

DEVELOPMENTAL KIDNEY DISEASES

(DISEASES THAT ARE INHERITED OR OCCUR DURING DEVELOPMENT OF THE KIDNEY)



BASICS

OVERVIEW

Congenital (existing at birth) and developmental kidney diseases are a group of diseases in which the kidney may be abnormal in appearance or may be abnormal in its ability to function normally or both. These diseases result from inherited or genetic problems or disease processes that affect the development and growth of the kidney before or shortly after birth. Examples of congenital and developmental kidney diseases include the following:

- Failure of kidney formation (known as “renal agenesis”)—complete absence of one or both kidneys; one or both kidneys failed to form
- Abnormal kidney development (known as “renal dysplasia”)—disorganized or abnormal kidney tissue development
- Displacement of one or both kidneys (known as “renal ectopia”)—one or both kidneys are located in an abnormal position in the body at birth (congenital); ectopic kidneys may be fused together
- Glomerulopathy—a group of small blood vessels in the functional unit of the kidney is the glomerulus; “glomerulopathy” is the broad name for any type of glomerular disease
- Kidney disease involving the tubules and tissue spaces (known as “tubulointerstitial nephropathy”)—a noninflammatory disorder of kidney tubules and tissue spaces (known as “interstitium”)
- Polycystic kidney disease—characterized by formation of multiple, variable-sized cysts throughout the kidney tissue
- Dilation of small blood vessels in the kidney (known as “renal telangiectasia”)—characterized by multiple blood-vessel malformations involving the kidneys and other organs
- Amyloidosis of the kidney—(amyloidosis is a group of conditions of differing cause in which insoluble proteins [amyloid] are deposited outside of cells in various tissues and organs, compromising their normal function); specifically, amyloidosis of the kidney is the deposition of amyloid outside the cells of the blood vessels in the glomerulus (glomerular capillaries), the glomerulus itself, and the tissue spaces (known as “interstitium”)
- Nephroblastoma—a congenital kidney tumor
- Multifocal renal-cyst adenocarcinoma—a hereditary kidney cancer in dogs
- Fanconi’s syndrome—a generalized functional abnormality involving the tubules of the kidney, characterized by impaired reabsorption of glucose, phosphate, electrolytes, amino acids, and uric acid
- Presence of glucose in the urine due to primary kidney disease (known as “primary renal glucosuria”)—an isolated functional defect in the reabsorption of glucose by the kidney tubules, characterized by the presence of glucose (sugar) in the urine when the animal has normal blood glucose levels
- Cystinuria—excessive excretion of cystine (an amino acid) into the urine, caused by an isolated functional defect in the reabsorption of cystine and other dibasic amino acids by the kidney tubules
- Xanthinuria—excessive excretion of xanthine into the urine, caused by a deficiency in an enzyme, xanthine oxidase, and impaired conversion of hypoxanthine to xanthine and of xanthine to uric acid
- Hyperuricuria—excessive excretion of uric acid, sodium urate, or ammonium urate into the urine, caused by impaired conversion of uric acid to allantoin by the liver and enhanced excretion of uric acid by the kidney tubules
- Primary hyperoxaluria—a disorder characterized by intermittent high levels of oxalic acid or oxalates in the urine (known as “hyperoxaluria”), l-glyceric aciduria, oxalate kidney disease (nephropathy), and acute kidney failure
- Congenital nephrogenic diabetes insipidus—“water diabetes”—a disorder of kidney concentrating ability, caused by diminished kidney responsiveness to antidiuretic hormone, such that the kidney does not reabsorb water and excessive urine is produced

GENETICS

Familial kidney disorders have been reported in the following dogs and cats:

- Renal agenesis in beagles and Doberman pinschers
- Renal dysplasia in Alaskan malamutes, boxers, chow chows, golden retrievers, keeshonden, Lhasa apsos, miniature schnauzers, shih tzu, soft-coated wheaten terriers, and standard poodles
- Glomerulopathy in beagles, Bernese mountain dogs, Brittanys, bullmastiffs, bull terriers, Doberman pinschers, English cocker spaniels, Newfoundlands, Pembroke Welsh corgis, rottweilers, samoyeds, and soft-coated wheaten terriers
- Tubulointerstitial nephropathy in Norwegian elkhounds
- Polycystic renal disease in the following breeds of dog: beagle, bull terrier, Cairn terrier, West Highland white terrier and in the following breeds of cat: Persian, exotic shorthair, and Himalayan
- Renal telangiectasia in Pembroke Welsh corgis
- Renal amyloidosis in the following breeds of cat: Abyssinian, oriental shorthair, and Siamese and in the following breeds of dog: English foxhound and Chinese shar pei
- Renal cystadenocarcinoma in German shepherd dogs
- Fanconi’s syndrome in basenjis and border terriers
- Primary renal glucosuria in Norwegian elkhounds
- Cystinuria in the following breeds of dog: basset hound, English bulldog, dachshund, French bulldog, Irish terrier, mastiff, Newfoundland,

Scottish deer hound, Staffordshire bull terrier, and Australian cattle dog and in the following breed of cat: domestic

- Xanthinuria in Cavalier King Charles spaniels
- Hyperuricuria in Dalmatians and English bulldogs
- Primary hyperoxaluria in the following breed of dog: Tibetan spaniel and in the following breed of cat: domestic shorthair

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Sporadic cases of congenital/developmental kidney disease can occur without a familial predisposition in any breed of dog or cat
- For familial kidney disorders, see **GENETICS**

Mean Age and Range

- Most patients are less than 5 years of age at time of diagnosis

Predominant Sex

- Familial cystinuria occurs primarily in male dogs; both sexes are affected in Newfoundlands
- Samoyed hereditary glomerulopathy is more common in males than females; both sexes are affected in Newfoundlands
- Familial glomerulonephropathy of Bernese mountain dogs is more common in females than males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs are related to chronic kidney failure, such as lack of appetite (anorexia), sluggishness (lethargy), excessive urination (polyuria) and excessive thirst (polydipsia), weight loss, vomiting
- Some glomerulopathies are associated with abdominal distension, fluid build-up (edema), or other signs of the nephrotic syndrome (medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin)
- Abdominal distension in some patients with polycystic kidneys or kidney cancer
- Bloody urine (hematuria) in some patients with renal telangiectasia or kidney cancer
- Apparent abdominal pain in some patients with renal telangiectasia
- Patients with one-sided lack of development of a kidney (renal agenesis), misplaced (ectopic) kidneys, and isolated kidney tubular transport defects are frequently asymptomatic
- Fluid build-up in the abdomen (known as “ascites”) or fluid build-up under the skin (known as “pitting edema”) in some patients with protein-losing glomerulopathies (a group of kidney diseases where excessive amounts of protein are lost through the urine) or amyloidosis (a group of conditions of varied cause in which insoluble proteins [amyloid] are deposited outside of the cells in various tissues and organs, compromising their normal function)
- Enlarged kidney (known as “renomegaly”) or abdominal mass lesions may be identified in some patients with polycystic kidneys, kidney cancer, or fused ectopic kidneys
- Renal pain may be seen in some patients with renal telangiectasia

CAUSES

Nonhereditary

- Infectious agents—feline panleukopenia virus and canine herpesvirus infection have been associated with renal dysplasia
- Drugs—corticosteroids, diphenylamine, and biphenyls have been associated with polycystic kidneys; chlorambucil and sodium arsenate have been associated with renal agenesis
- Dietary factors—too low levels of vitamin A (hypovitaminosis A) or too high levels of vitamin A (hypervitaminosis A) have been associated with renal ectopia

RISK FACTORS

- See factors listed under **CAUSES**



TREATMENT

HEALTH CARE

- The nature of congenital and developmental kidney disorders often prevents specific treatment
- Supportive or symptomatic treatment may improve quality of life and minimize progression in patients with kidney dysfunction
- Treatment options are based on clinical signs and laboratory diagnostic testing
- Specific treatment is determined after diagnosing the type of kidney disease or clinical syndrome

DIET

- Patients with chronic kidney failure—restrict phosphorus and moderately restrict protein
- Patients with high blood pressure (hypertension)—restrict sodium



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Specific treatment is determined by diagnosing the type of kidney diseases or clinical syndrome



FOLLOW-UP CARE

PATIENT MONITORING

- Specific monitoring is determined by diagnosing the type of kidney diseases or clinical syndrome
- Blood work (such as complete blood count, serum biochemistry profile) and urine testing (such as urinalysis, microalbuminuria testing, urine protein:creatinine [UP/C] ratio)

PREVENTIONS AND AVOIDANCE

- Congenital and developmental kidney disorders are irreversible, so control lies in preventing breeding of affected animals
- Always consider early identification and correction of predisposing factors (genetic and non-genetic) that may affect future offspring

POSSIBLE COMPLICATIONS

- Acute or chronic kidney failure
- Nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin)
- Presence of stones in the urinary tract (known as “urolithiasis”)
- Dilation of the cup-shaped cavity or pelvis of the kidney due to blockage of the flow of urine (known as “hydronephrosis”)
- Urinary tract infection

EXPECTED COURSE AND PROGNOSIS

- Highly variable; depends on the specific disorder, the extent of primary lesions, and the severity of kidney dysfunction
- Most congenital and developmental disorders are irreversible and may result in advanced chronic kidney failure, but some patients with mild-to-moderate kidney dysfunction may remain stable for long periods
- Patients with some disorders may remain asymptomatic, unless the disorder is complicated by development of urinary stones (urolithiasis), urinary tract infection, or other disease processes that promote progressive kidney dysfunction



KEY POINTS

- Congenital (existing at birth) and developmental kidney diseases are a group of diseases in which the kidney may be abnormal in appearance or may be abnormal in its ability to function normally or both
- These diseases result from inherited or genetic problems or disease processes that affect the development and growth of the kidney before or shortly after birth
- Congenital and developmental kidney disorders are irreversible, so control lies in preventing breeding of affected animals
- Supportive or symptomatic treatment may improve quality of life and minimize progression in patients with kidney dysfunction

KIDNEY FAILURE—SUDDEN (ACUTE) UREMIA



BASICS

OVERVIEW

- “Uremia” is the medical term for excessive levels of urea and other nitrogenous waste products in the blood (it is also known as “azotemia”)
- Sudden (acute) uremia is a clinical presentation characterized by sudden onset of kidney failure; urea and other nitrogenous waste products build up, leading to clinical signs
- It is potentially reversible, if diagnosed quickly and treated aggressively
- The kidney filters the blood and removes various waste products from the body as it produces urine; the kidney is involved in maintaining the normal fluid volume of the body; each kidney is composed of thousands of nephrons (the functional units of the kidney, each consisting of the glomerulus [a tuft of blood capillaries—the “blood filter”] and a series of tubes and ducts, through which the filtered fluid flows, as urine is produced)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Peak incidence in dogs and cats—6 to 8 years of age
- Older animals at greater risk

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden onset of lack of appetite (known as “anorexia”); listlessness; depression; vomiting (with or without the presence of blood); diarrhea (with or without the presence of blood); bad breath (known as “halitosis”); wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”); seizures; and production of only small amounts of urine (known as “oliguria”)/or no urine (known as “anuria”) or increased volume of urine (known as “polyuria”)
- Observed poison or drug exposure; recent medical or surgical conditions
- Normal body condition and hair coat; dehydration (or overhydration due to administration of fluids); ulcers in the mouth; inflammation of the tongue (known as “glossitis”) and death of tissues of the tongue; low body temperature (known as “hypothermia”) or fever; rapid breathing (known as “tachypnea”); slow heart rate (known as “bradycardia”); inability to feel the urinary bladder during physical examination; and enlarged, painful, firm kidneys

CAUSES

Causes Related to Abnormal Circulation to the Kidneys

- Shock; trauma; blood clots (known as “thromboembolism”); heat stroke; excessive narrowing of the blood vessels (known as “vasoconstriction,” such as following the administration of nonsteroidal anti-inflammatory drugs [NSAIDs]); adrenal gland insufficiency; excessive enlargement or dilation of blood vessels (known as “vasodilation,” such as following the administration of angiotensin-converting enzyme [ACE] inhibitors or medications to decrease blood pressure [antihypertensive drugs]); prolonged anesthesia; heart failure

Compounds/Medications that are Toxic to the Kidneys

- Ethylene glycol (found in antifreeze); antibiotics (aminoglycosides); antifungal medications (amphotericin B); chemotherapeutic agents (such as cisplatin and doxorubicin); nonsteroidal anti-inflammatory drugs (NSAIDs); radiographic contrast agents; heavy metals (such as lead, mercury, arsenic, thallium); insect or snake venom; calcium; grape or raisin ingestion (dogs); and lily ingestion (cats)

Generalized Disease Affecting the Kidneys

- Infectious disease (such as leptospirosis or Lyme disease); immune-mediated disease (such as inflammation and accompanying dysfunction of glomeruli [plural of glomerulus] of the kidney [known as “glomerulonephritis”] and inflammation of the arteries [known as “arteritis”]); inflammation of the pancreas (known as “pancreatitis”); generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”); blood-clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”); liver failure; heat stroke; blood transfusion reactions; bacterial inflammation/infection of the lining of the heart (known as “bacterial endocarditis”); bacterial infection/inflammation of the kidney (known as “pyelonephritis”); and cancer (such as lymphoma; “lymphoma” is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body)
- Blockage or obstruction of one or both ureters (the tubes running from the kidneys to the bladder) in cats

RISK FACTORS

- Pre-existing long-term (chronic) kidney disease; dehydration; generalized bacterial infection (known as “sepsis”); low blood volume (known as “hypovolemia”); low blood pressure (known as “hypotension”); advanced age; coexistent disease; low levels of sodium in the blood (known as “hyponatremia”); low levels of potassium in the blood (known as “hypokalemia”); low levels of calcium in the blood (known as “hypocalcemia”); and acidosis (a condition in which levels of acid are increased in the blood)
- Medications (such as the diuretic, furosemide; nonsteroidal anti-inflammatory drugs [NSAIDs]; angiotensin-converting enzyme [ACE] inhibitors; antibiotics [aminoglycosides]); prolonged anesthesia; acidifying diets; trauma; multiple organ disease; and high environmental temperature



TREATMENT

HEALTH CARE

- Inpatient management; eliminate inciting insults; discontinue kidney toxic drugs; establish and maintain circulation and blood flow; treat life-threatening fluid imbalances, biochemical abnormalities, and uremic toxicities
- If a poison is the likely cause of sudden (acute) uremia and kidney failure, follow appropriate treatment for the specific poison (treatment may include inducing vomiting, flushing of the stomach [known as “gastric lavage”], and administering activated charcoal and specific antidotes); early hemodialysis (procedure to remove waste products from the blood) can eliminate poisons
- Low blood volume (hypovolemia) or dehydration—correct estimated fluid deficits with normal (0.9%) saline or balanced fluids within 2 to 4 hours; blood losses may be replaced by blood transfusion; once the patient is hydrated, ongoing fluid requirements are provided; avoid overhydration
- High blood volume (known as “hypervolemia”) or overhydration—stop fluid administration and eliminate excess fluid by treatment with medications to remove excess fluid from the body (known as “diuretics”) or dialysis (procedure to remove waste products from the body)

DIET

- Restrict intake of food and water by mouth until vomiting subsides
- Fat and protein stores will be consumed by the body while patient is not eating (anorexia); resting energy requirements must be provided by 3 to 5 days, using moderately protein-restricted diets or feeding special solutions formulated to control excessive levels of urea and other nitrogenous waste products in the blood (uremia or azotemia) and supply caloric requirements
- Nutrition may be provided by intravenous feeding (known as “parenteral nutrition”) in vomiting animals
- Tube feeding may be provided for animals that are not eating (anorexia) and are not vomiting—caloric and protein requirements may be supplied by using kidney diets that have been liquefied in a kitchen-type blender, special liquid diets, or formulated diets

SURGERY

- Surgery may be required for sudden (acute) blockage or obstruction of one or more ureters (the tubes running from the kidneys to the bladder) in cats
- Kidney transplant may provide long-term survival for cats with severe, sudden (acute) kidney injury



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Inadequate Urine Production

- Ensure patient has normal blood volume
- Administer medications to remove excess fluid from the body (diuretics), such as mannitol or furosemide (alternative or subsequent to mannitol)
- If diuretic treatments fail to induce increased production of urine within 4 to 6 hours, consider dialysis (procedure to remove waste products from the body)

Acid–Base Disorders

- Bicarbonate

Vomiting

- No food or water (nothing by mouth) until vomiting subsides
- Reduce stomach-acid production—famotidine or ranitidine or omeprazole

- Medication to protect the lining of the stomach (known as a “mucosal protectant”)—sucralfate
- Medications to control nausea and/or vomiting (known as “antiemetics”)—metoclopramide, ondansetron, or dolasetron
- Control of vomiting—chlorpromazine, prochlorperazine, acepromazine

Peritoneal Dialysis or Hemodialysis

- “Peritoneal dialysis” is a type of dialysis in which fluids are put into the abdomen and the lining of the abdomen (known as the “peritoneum”) acts as a filter to remove waste products from the blood—after a certain amount of time, the fluids and waste products are removed from the abdomen; “hemodialysis” is a procedure to remove waste products from the blood
- Dialysis can stabilize the patient until kidney function is restored or until corrective surgery is performed; without dialysis, most patients that produce only small amounts of urine (oliguria) die before kidney repair can occur



FOLLOW-UP CARE

PATIENT MONITORING

- Fluid, electrolyte, and acid–base balances; body weight; blood pressure; urine output; and clinical status—monitor daily

PREVENTIONS AND AVOIDANCE

- Anticipate the potential for sudden (acute) kidney injury in aged patients or those with generalized (systemic) disease, generalized bacterial infection (sepsis), trauma, receiving kidney toxic drugs, multiple organ failure, or those undergoing prolonged anesthesia; maintenance of hydration, mild saline volume expansion, and administration of mannitol may be preventive
- Monitor urine production and increased levels of urea and other nitrogenous waste products (azotemia) in high-risk patients

POSSIBLE COMPLICATIONS

- Seizures; coma; irregular heart beats (known as “cardiac arrhythmias”); increased blood pressure (hypertension); congestive heart failure; fluid build-up in the lungs (known as “pulmonary edema”); inflammation of the lungs due to the presence of urea and other nitrogenous waste products (known as “uremic pneumonitis”); aspiration pneumonia; bleeding in the gastrointestinal tract; shock due to low blood volume; generalized bacterial infection (sepsis); stopping of the heart and breathing (known as “cardiopulmonary arrest”); and death

EXPECTED COURSE AND PROGNOSIS

- Prognosis depends on cause, extent of damage, coexistent diseases, and multiple organ involvement
- Infectious and obstructive causes have a better prognosis for recovery than toxic causes



KEY POINTS

- Poor prognosis for complete recovery
- Potential for complications of treatment (such as fluid overload, generalized bacterial infection [sepsis], and multiple organ failure)
- Prolonged hospitalization and treatment is expensive
- Alternatives to conventional medical management are available; they include peritoneal dialysis or hemodialysis (procedures to remove waste products from the blood) and kidney transplantation

LONG-TERM (CHRONIC) KIDNEY FAILURE



BASICS

OVERVIEW

- Long-term (chronic) kidney failure is defined as the pet having an excess level of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) in combination with a urine specific gravity of less than 1.030 in dogs and less than 1.035 in cats; “urine specific gravity” measures the concentration of substances (such as salt, glucose, protein) in the urine—the higher the specific gravity, the higher the concentration of particles and the lower amount of water in the urine; the kidneys remove water from the urine to maintain hydration levels—as kidney function decreases, the kidney is less able to remove water from the urine and the urine specific gravity drops; urine specific gravity provides information regarding the kidney’s ability to conserve fluid (concentrate urine) or to remove excess fluid from the body (dilute urine); urine specific gravity fluctuates normally based on the amount the pet drinks and the amount of salt in the diet
- Long-term (chronic) kidney failure results from primary kidney disease that has persisted for months to years; characterized by irreversible loss of kidney function, which tends to deteriorate progressively over months to years
- The kidney filters the blood and removes various waste products from the body as it produces urine; the kidney is involved in maintaining the normal fluid volume of the body; each kidney is composed of thousands of nephrons (the functional units of the kidney, each consisting of the glomerulus [a tuft of blood capillaries—the “blood filter”] and a series of tubes and ducts, through which the filtered fluid flows, as urine is produced)

GENETICS

Inherited in the following breeds (mode of inheritance, known or suspected, indicated in parentheses):

- Abyssinian cats (autosomal dominant with incomplete penetrance)
- Persian cats (autosomal dominant)
- Bull terrier (autosomal dominant)
- Cairn terrier (autosomal recessive)
- German shepherd dog (autosomal dominant)
- Samoyed (X-linked dominant)
- English cocker spaniel (autosomal recessive)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- All breeds of dogs and cats are affected
- Familial (runs in certain families or lines of animals) kidney disease resulting in long-term (chronic) kidney failure has been reported in the basenji, beagle, bull terrier, Cairn terrier, chow chow, Doberman pinscher, English cocker spaniel, German shepherd dog, golden retriever, Lhasa apso, miniature schnauzer, Norwegian elkhound, rottweiler, Samoyed, Chinese shar pei, shih tzu, soft-coated wheaten terrier, and standard poodle, and in Abyssinian cats

Mean Age and Range

- Mean age at diagnosis is approximately 7 years in dogs and 9 years in cats
- Animals of any age can be affected, but number of affected animals is higher with increasing age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs are related to the severity of kidney dysfunction and the presence or absence of complications (such as high blood pressure [known as “hypertension”])
- Cats with mild, long-term (chronic) kidney failure may not have any clinical signs
- An animal with stable, long-term (chronic) kidney failure may no longer be able to offset the decreased kidney function, resulting in a medical crisis (so called “uremic crisis”)
- Increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)—less frequent in cats than dogs
- Lack of appetite (known as “anorexia”)
- Sluggishness (lethargy)
- Vomiting
- Weight loss

- Urinating at night (known as “nocturia”)
- Constipation
- Diarrhea
- Degeneration of the retina (back part of the eye) due to high blood pressure (known as “hypertensive retinopathy”) and resulting sudden (acute) blindness
- Seizures or coma—late
- Cats also may have excessive drooling (known as “ptyalism”) and muscle weakness with abnormal position of the head and neck, in which the chin is located near the chest (known as “neck ventroflexion”), because of a muscle disorder caused by low levels of potassium in the blood (known as “hypokalemic myopathy”)
- Small, irregular kidneys (or enlarged kidneys secondary to polycystic kidney disease [disease characterized by multiple fluid-filled sacs or cysts in the kidney] or lymphoma [a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body])
- Dehydration
- Extreme weight loss with muscle wasting (known as “cachexia”)
- Pale gums and moist tissues of the body
- Ulceration of the mouth
- Abnormal odor to the mouth/breath due to excessive levels of urea and other nitrogenous waste products in the blood (uremia or azotemia; abnormal odor known as “uremic breath odor”)
- Constipation
- Bone disorder caused by long-term (chronic) kidney failure leading to abnormal levels of calcium and phosphorus in the blood and resulting loss of mineralization of the bones (known as “renal osteodystrophy”)

CAUSES

- Most cases are of unknown cause (so called “idiopathic disease”), and the disease is termed “chronic generalized nephropathy” (“nephropathy” is any disease of the kidney)
- Causes include familial (runs in certain families or lines of animals) and congenital (present at birth) kidney disease; exposure to products that are toxic to the kidneys; increased levels of calcium in the blood (known as “hypercalcemia”); low levels of potassium in the blood (known as “hypokalemia,” condition known as “hypokalemic nephropathy”); inflammation and accompanying dysfunction of glomeruli (plural of glomerulus) of the kidney (known as “glomerulonephritis”); amyloidosis (a group of conditions in which insoluble proteins [known as “amyloid”] are deposited outside the cells in the kidney, compromising normal function); infection/inflammation of the kidney (known as “pyelonephritis”); polycystic kidney disease (multiple fluid-filled sacs or cysts in the kidney); kidney stones (known as “nephroliths”); long-term (chronic) urinary blockage or obstruction; drugs; cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body (lymphoma); leptospirosis (following sudden [acute] kidney failure), feline infectious peritonitis (FIP) in cats; and, possibly, diabetes mellitus (“sugar diabetes”)

RISK FACTORS

- Aging, increased levels of calcium in the blood (hypercalcemia), low levels of potassium in the blood (hypokalemia) in cats, high blood pressure (known as “hypertension”), urinary tract infection, diabetes mellitus (“sugar diabetes”)



TREATMENT

HEALTH CARE

- Patients that are able to offset the loss of function of the kidneys may be managed as outpatients
- Patients in uremic crisis (vomiting, dehydrated, “sick”) should be managed as inpatients—correct fluid and electrolyte deficits with intravenous fluid therapy (such as administration of lactated Ringer’s solution); correct dehydration over 6 to 12 hours to prevent additional kidney injury from poor or diminished blood flow (known as “ischemia”)
- Subcutaneous fluid therapy (daily or every other day) may benefit patients with moderate-to-severe long-term (chronic) kidney failure
- Hemodialysis (procedure to remove waste products from the blood) and kidney transplantation are available at selected veterinary referral hospitals

ACTIVITY

- Unrestricted

DIET

- Reduced dietary protein, phosphorus, and sodium with adequate buffering capacity (alkalinizing diet)

- Supplemental n-3 fatty acids may be beneficial
- Recent studies in dogs and cats indicate that compared to maintenance diets, feeding kidney diets delays onset of uremic crisis (signs of vomiting, dehydration, being “sick”) and kidney-related death
- Free access to fresh water at all times

SURGERY

- Avoid low blood pressure (known as “hypotension”) during anesthesia, to prevent additional kidney injury
- Kidney transplants have been performed successfully in dogs and cats with advanced disease



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Uremic Crisis (signs of vomiting, dehydration, and being “sick”)

- Famotidine to minimize nausea and vomiting
- Potassium chloride in intravenous fluids or potassium gluconate administered by mouth, as needed to correct low levels of potassium in the blood (hypokalemia)

Compensated Long-Term (Chronic) Kidney Failure (in which pet is able to offset loss of kidney function)

- Famotidine to minimize nausea
- Potassium gluconate administered by mouth, as needed to correct low levels of potassium in the blood (hypokalemia)
- Intestinal phosphate binders (such as aluminum carbonate, administered by mouth with meals), as needed to correct increased levels of phosphate in the blood (known as “hyperphosphatemia”)
- Calcitriol
- Erythropoietin (the hormone that stimulates the bone marrow to produce red-blood cells)
- Amlodipine or angiotensin-converting enzyme (ACE) inhibitors (such as enalapril or benazepril), as needed to treat high blood pressure (hypertension); amlodipine is more effective than ACE inhibitors in cats with long-term (chronic) kidney failure-induced hypertension; if pet does not respond to one drug, consider combination of amlodipine and ACE inhibitor with frequent monitoring of blood pressure

Other Drugs

- Metoclopramide can be used in addition to H₂-blockers (such as famotidine) to treat vomiting caused by excessive level of urea and other nitrogenous waste products in the blood (uremia or azotemia)
- H₂-blockers (ranitidine or cimetidine) may be used instead of famotidine for inflammation of the stomach caused by excess level of urea and other nitrogenous waste products in the blood (condition known as “uremic gastritis”)
- Ondansetron for cases that have nausea and vomiting that do not respond to other medical treatment



FOLLOW-UP CARE

PATIENT MONITORING

- Dogs and cats with long-term (chronic) kidney failure should be monitored at regular intervals, depending on therapy and severity of disease; initially weekly for patients receiving calcitriol or erythropoietin; re-evaluate patients with mild-to-moderate chronic kidney failure every 1 to 3 months

PREVENTIONS AND AVOIDANCE

- Do not breed animals with familial (runs in certain families or lines of animals) kidney disease

POSSIBLE COMPLICATIONS

- Generalized (systemic) high blood pressure (hypertension), inflammation of the mouth caused by excess level of urea and other nitrogenous waste products in the blood (condition known as “uremic stomatitis”), inflammation of the stomach and intestines (known as “gastroenteritis”), low levels of red-blood cells (known as “anemia”), secondary urinary tract infection

EXPECTED COURSE AND PROGNOSIS

- Short-term—depends on severity

- Long-term—guarded to poor because long-term (chronic) kidney failure tends to be progressive over months to years
- Tends to progress to terminal, long-term (chronic) kidney failure over months to years



KEY POINTS

- Tends to progress to terminal, long-term (chronic) kidney failure over months to years
- Carefully consider use of animals in breeding programs due to the genetic basis of familial (runs in certain families or lines of animals) kidney diseases; do not breed animals with familial kidney disease

CORNEAL AND SCLERAL LACERATIONS



BASICS

OVERVIEW

- “Corneal” refers to the cornea; “scleral” refers to the sclera of the eye
- The “cornea” is the clear outer layer of the front of the eye; the “sclera” is the white part of the eye, it is composed of a tough covering that protects the eyeball
- “Penetrating” injury—a wound or foreign body that enters, but does not completely pass through the cornea or sclera; it also is known as a “nonperforating” injury
- “Perforating” injury—a wound or foreign body that completely passes through the cornea or sclera; greater risk of vision loss than with penetrating injury
- “Simple”—involves only the cornea or sclera; may be penetrating or perforating; other eye structures are intact (that is, not injured)
- “Complicated”—a type of perforating injury; involves other eye structures in addition to the cornea or sclera; may involve the uvea (the entire middle layer of the eyeball that contains the blood vessels; it is composed of the iris [the colored or pigmented part of the eye], the ciliary body [the area between the iris and the choroid], and the choroid [located under the back part of the eye—the retina]; the vitreous (the clear, gel-like material that fills the back part of the eyeball [between the lens and the retina]); or the retina (back part of the eye that contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision); may also have trauma to the lens leading to cataracts or lacerations to the eyelids
- The pupil is the circular or elliptical opening in the center of the iris of the eye; light passes through the pupil to reach the back part of the eye (known as the “retina”); the iris is the colored or pigmented part of the eye—it can be brown, blue, green, or a mixture of colors

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Varies with tissues affected
- Usually sudden (acute) onset of signs
- Often a history of running through heavy vegetation, being hit by gunshot pellets or other projectiles, or being scratched by a cat
- Trauma itself may not be observed
- Common—corneal, scleral, or eyelid deformity; fluid build-up (known as “edema”) in affected tissues; bleeding (hemorrhage)
- May see a retained foreign body
- Lacerations often rapidly seal; may appear only as a blood-filled mass under the moist tissues of the eye (known as a “subconjunctival hematoma”)
- May also see iris defects; pupil distortion; blood in the anterior chamber of the eye (the front part of the eye, between the cornea and the iris; accumulation of blood known as “hyphema”); cataract (opacity in the normally clear lens, preventing passage of light to the back part of the eye [retina]); bleeding in the vitreous (the clear, gel-like material that fills the back part of the eyeball); separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid;” condition known as “retinal detachment”); and protrusion of the eyeball (known as “exophthalmia”)

CAUSES

- Blunt or sharp trauma

RISK FACTORS

- Pre-existing visual impairment
- Young, naive, or highly excitable animals
- Hunting or running through heavy vegetation
- Fighting



TREATMENT

HEALTH CARE

- Depends on severity
- Outpatient—if integrity of the eyeball is ensured
- Sedation—consider for excited or fractious patients
- When walking—apply an Elizabethan collar and use a harness to avoid increasing pressure in the affected eye

Injuries Considered for Medical Treatment

- Penetrating or nonpenetrating wounds with no overriding or gaping of the wound edges—apply an Elizabethan collar; administer antibiotic and/or atropine eye solutions directly to the eye (“topical treatment”)
- Penetrating or nonpenetrating wounds with mild gaping of wound edges or shelved edges—apply a therapeutic soft contact lens and an Elizabethan collar; administer antibiotic and/or atropine eye solutions directly to the eye (“topical treatment”)
- Simple full-thickness, pinpoint corneal perforation with a formed anterior chamber and no uveal prolapse—sedentary patients; use a therapeutic soft contact lens and an Elizabethan collar; apply topical antibiotic and/or atropine ophthalmic solutions; re-examine a few hours after applying the lens and at 24 and 48 hours

ACTIVITY

- Usually confined indoors (cats) or limited to leash walks until healing is complete

SURGERY

Injuries Requiring Surgical Exploration or Repair

- Full-thickness corneal or scleral lacerations
- Complicated injuries
- Suspected retained foreign body or a scleral rupture involving the back of the eye
- Simple, penetrating or nonpenetrating wound with edges that are moderately or overtly gaping and that are long or more than two-thirds the corneal thickness

Injuries Considered for Surgical Exploration or Repair

- Small, simple, full-thickness corneal lacerations
- Large conjunctival lacerations
- Partial-thickness corneal or scleral lacerations in an active patient



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antibiotics

- Complicated wounds, those with retained plant material, and those caused by blunt trauma with tissue devitalization—infection common
- Bacterial infection/inflammation of the tissues within the eyeball (known as “endophthalmitis”)—seen in 5% to 7% of animals with perforations; very rare in penetrating wounds
- Penetrating wound—topical (applied to the eye) antibiotics alone (such as neomycin, polymyxin B, and bacitracin) or gentamicin solution usually sufficient
- Perforating wounds—systemic (administered by injection or by mouth) ciprofloxacin (dogs); topical (applied to the eye) cefazolin and fortified gentamicin or tobramycin
- Topical (applied to the eye) ciprofloxacin eye solution—may be used instead of the combination of topical cefazolin and a fortified aminoglycoside

Anti-inflammatory Drugs

- Topical (applied to the eye) 1% prednisolone acetate or 0.1% dexamethasone solution—as soon as the wound is sutured or the surface is healing, if no infection is present
- Systemic (administered by mouth) prednisone—for sutured or healing wounds when inflammation is severe; when the lens or more posterior structures are involved; when the wound is infected or not healing and control of inflammation is mandatory to preserve the eye
- Topical (applied to the eye) nonsteroidal anti-inflammatory drugs (NSAIDs)—suprofen or flurbiprofen; may be used if topical steroids are not indicated (for example, infection is present) and control of inflammation is mandatory to preserve the eye

Medications to Dilate the Pupil (Known as “Mydriatics”) and to Relieve Eye Pain

- 1% atropine eye solution—when significant constriction of the pupil (known as “miosis”) is present or inflammation is identified in the front chamber of the eye (known as “anterior chamber reaction”)

Medications to Relieve Pain (Analgesics)

- Topical atropine or oral aspirin (dogs)—may provide sufficient pain relief; use analgesics only as directed by your pet’s veterinarian
- Butorphanol—dogs and cats; for mild pain
- Oxymorphone—dogs and cats; for sudden (acute) severe pain; when sedation is required



FOLLOW-UP CARE

PATIENT MONITORING

- Deep or long penetrating wounds that have not been sutured and perforating wounds—recheck every 24 to 48 hours for the first several days to ensure integrity of the eyeball, to monitor for infection, and to check control of eye inflammation
- Superficial penetrating wounds—usually rechecked at 3- to 5-day intervals, until healed
- Antibiotic therapy—altered according to bacterial culture and sensitivity results

PREVENTIONS AND AVOIDANCE

- Take care when introducing new puppies to households with cats that have front claws
- Minimize running through dense vegetation
- Consider having a bottle of saline eyewash to irrigate or flush debris from the eye
- Minimize visually impaired or blind dogs' exposure to dense vegetation

POSSIBLE COMPLICATIONS

- Loss of the eye or vision
- Long-term (chronic) eye inflammation or pain
- Post-traumatic sarcoma, a type of cancer, may develop in blind cat eyes that have been injured severely; consider surgical removal of the eyeball and associated tissues (known as “enucleation”) for all blind, injured feline eyes to prevent post-traumatic sarcoma

EXPECTED COURSE AND PROGNOSIS

- Most eyes with corneal lacerations or having a retained corneal foreign body are salvageable
- The further back the injury is located in the eye, the poorer the prognosis for retention of vision
- Poor prognosis—involvement of the sclera (the white part of the eye, it is composed of a tough covering that protects the eyeball) or uvea (the entire middle layer of the eyeball that contains the blood vessels); no light perception; perforating injuries involving the lens; significant bleeding into the vitreous (the clear, gel-like material that fills the back part of the eyeball); or separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)
- Penetrating injuries usually better prognosis than perforating injuries
- Blunt trauma carries a poorer prognosis than sharp trauma



KEY POINTS

- The full extent of the injury (cataracts, retinal detachment, infection) may not be apparent until several days or weeks after the injury; therefore, long-term follow-up is necessary

LAMENESS IN DOGS



BASICS

OVERVIEW

- “Lameness” is a clinical sign
- A disturbance in gait and locomotion in response to pain, injury, or abnormal anatomy

GENETICS

- Depends on specific disease

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Depend on specific disease

Mean Age and Range

- Depend on specific disease

Predominant Sex

- Depends on specific disease

SIGNS/OBSERVED CHANGES in the ANIMAL

- Lameness—may involve one or more limbs; varies in severity from subtle lameness to non-weightbearing (that is, carrying the leg)
- Lameness may be better or worse after rest or after activity
- If only one forelimb is involved—head and neck moves upward when the affected limb is placed on the ground and drops when the sound limb bears weight
- If only one hind limb is involved—pelvis drops when affected leg bears weight, rises when weight is lifted
- If both hind limbs are involved—forelimbs are carried lower to shift weight forward
- Nervous system signs
- Posture may be abnormal when standing, getting up or laying down, or sitting
- Abnormal gait may be seen when walking, trotting, climbing stairs, or doing figure-eights
- May have loss of muscle mass (known as “muscle atrophy”)
- Bones and/or joints may be abnormal
- Decreased range of motion
- Pain
- Grating detected with joint movement (known as “crepitus”)

CAUSES

Forelimb Lameness

Growing Dog (Less than 12 Months of Age)

- Osteochondrosis of the shoulder
- Shoulder dislocation (luxation) or partial dislocation (subluxation)—congenital (present at birth)
- Osteochondrosis of the elbow
- Ununited anconeal process
- Fragmented medial coronoid process
- Elbow incongruity
- Avulsion or calcification of the flexor muscles—elbow
- Asymmetric growth of the radius and ulna (bones of the foreleg)
- Panosteitis
- Hypertrophic osteodystrophy
- Trauma—soft tissue; bone; joint

- Infection—local; generalized (systemic)
- Nutritional imbalances
- Congenital (present at birth) abnormalities

Mature Dog (Greater than 12 Months of Age)

- Degenerative joint disease (progressive and permanent deterioration of joint cartilage)
- Bicipital tenosynovitis
- Calcification or mineralization of supraspinatus or infraspinatus tendon
- Contracture of supraspinatus or infraspinatus muscle
- Soft-tissue or bone cancer—primary; metastatic (cancer that has spread)
- Trauma—soft tissue; bone; joint
- Panosteitis
- Polyarthropathies
- Polymyositis
- Polyneuritis

Hindlimb

Growing Dog (Less than 12 Months of Age)

- Hip dysplasia
- Avascular necrosis of femoral head—Legg-Calvé-Perthes disease
- Osteochondritis of stifle
- Patella luxation—medial or lateral
- Osteochondritis of hock
- Panosteitis
- Hypertrophic osteodystrophy
- Trauma—soft tissue; bone; joint
- Infection—local; generalized (systemic)
- Nutritional imbalances
- Congenital (present at birth) abnormalities

Mature Dog (Greater than 12 Months of Age)

- Degenerative joint disease (progressive and permanent deterioration of joint cartilage), secondary to hip dysplasia
- Cruciate ligament disease
- Avulsion of long digital extensor tendon
- Soft-tissue or bone cancer—primary; metastatic (cancer that has spread)
- Trauma—soft tissue; bone; joint
- Panosteitis
- Polyarthropathies
- Polymyositis
- Polyneuritis

RISK FACTORS

- Breed (size)
- Overweight
- Strenuous activity



TREATMENT

HEALTH CARE

- Depends on underlying cause

ACTIVITY

- Depends on underlying cause

DIET

- Depends on underlying cause
- Reducing diet, if dog is overweight or obese to decrease stress on joints

SURGERY

- Depends on underlying cause



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Pain relievers (known as “analgesics”) and nonsteroidal anti-inflammatory drugs (NSAIDs)—minimize pain; decrease inflammation; NSAIDs include such drugs as meloxicam, carprofen, etodolac, and deracoxib
- Steroids may be used in certain cases
- Medications intended to slow the progression of arthritic changes and protect joint cartilage (known as “chondroprotective drugs”), such as polysulfated glycosaminoglycans, glucosamine, and chondroitin sulfate—may help limit cartilage damage and degeneration; may help alleviate pain and inflammation



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on underlying cause

PREVENTIONS AND AVOIDANCE

- Depend on underlying cause

POSSIBLE COMPLICATIONS

- Depend on underlying cause

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause



KEY POINTS

- “Lameness” is a clinical sign
- A disturbance in gait and locomotion in response to pain, injury, or abnormal anatomy

LARYNGEAL DISEASE

(DISEASE OF THE VOICE BOX OR LARYNX)



BASICS

OVERVIEW

- The voice box or larynx serves as a passage for airflow from the external environment to the lungs; it protects the lungs from aspiration during swallowing and regurgitation; and it allows vocalization (such as barking or meowing)
- Laryngeal disease refers to any condition that alters normal structure and/or function of the voice box or larynx

GENETICS

- Paralysis (dogs)—inherited disorder in Bouvier des Flandres (inherited as an autosomal dominant trait); genetic susceptibility is suspected, but unproven, in Siberian huskies and bull terriers; paralysis of the voice box or larynx as part of a condition involving multiple nerves throughout the body (known as “laryngeal paralysis–polyneuropathy complex”) in young Dalmatians and rottweilers is considered to be inherited, but presently genetic basis is unproven

SIGNALMENT/DESCRIPTION OF ANIMAL

Species

- Dogs and cats
- Cats—incidence, based on limited reports in the literature, appears to be significantly lower than in dogs

Breed Predispositions

- Hereditary paralysis of the voice box or larynx in dogs—Bouvier des Flandres, Siberian huskies, mixed-breed huskies; and probably bull terriers; part of a generalized disorder involving several nerves (known as “polyneuropathy syndrome”)—Dalmatians, probably rottweilers
- Acquired (condition that develops sometime later in life/after birth) paralysis of the voice box or larynx in dogs—overrepresented in giant-breed dogs (St. Bernards, Newfoundlands) and large-breed dogs (Irish setters, Labrador retrievers, golden retrievers)
- Cats—no defined breed susceptibility

Mean Age and Range

- Hereditary paralysis of the voice box or larynx in dogs—onset of signs varies in the different breeds: Bouvier des Flandres—4 to 6 months of age; Dalmatians—4 to 8 months of age; rottweilers—11 to 13 weeks of age; white-coated German shepherd dogs—4 to 6 months of age
- Acquired (condition that develops sometime later in life/after birth) paralysis of the voice box or larynx in dogs—onset of signs seen at 1 to 12 years of age; reported mean, 9 to 12 years of age
- Cats—usually older, but seen occasionally in younger cats secondary to trauma or surgical procedures; median age in one report was 11 years of age
- Cancer: middle-aged to old dogs and cats

Predominant Sex

- Hereditary paralysis of the voice box or larynx—reported incidence in medical literature varies from 3:1 male-to-female ratio (that is, males are three times as likely to have hereditary voice-box paralysis than females) down to a 1:1 male-to-female ratio (that is, males and females are equally likely to have hereditary voice-box paralysis)
- Acquired (condition that develops sometime later in life/after birth) paralysis of the voice box or larynx in dogs—reported incidence of 2:1 male-to-female ratio (that is, males are twice as likely to have acquired voice-box paralysis than females)
- Acquired paralysis of the voice box or larynx in cats—reported incidence of approximately 1:1 male-to-female ratio (that is, males and females are approximately equal in likelihood to have acquired voice-box paralysis)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Directly related to the degree of impairment or restriction of airflow through the voice box or larynx
- Change in character of the bark or meow
- Occasional coughing
- Panting
- Reduced activity, exercise intolerance
- Abnormal breathing sounds with exertion or stress
- Signs associated with exertion, stress, or heat—severely difficult breathing; gagging and retching; vomiting; weakness and sluggishness (lethargy); collapse; even sudden death

- Noisy respiration and a high-pitched sound on inspiration (known as “stridor”)—most common
- Cats—inspiratory stridor less characteristic than in dogs
- Upper airway sounds are detected over the windpipe (trachea) and lungs, upon listening to the airways with a stethoscope
- If animal has aspiration pneumonia—short, sharp sounds (known as “crackles”) may be detected in small areas (localized) or in larger areas of the lungs; sounds are heard upon listening to the chest with a stethoscope
- Rectal temperature—usually elevated above normal, especially in warm weather

CAUSES

Paralysis

- Congenital—present at birth; inherited disorders
- Acquired (condition that develops sometime later in life/after birth)—most often of unknown cause (so called “idiopathic paralysis of the larynx”); vagal nerve abnormality (the vagus nerve supplies nerve fibers to the voice box [larynx], throat [pharynx], windpipe [trachea] and other organs); trauma to the neck; abnormality involving the recurrent laryngeal nerves (branches of the vagus nerve); diseases in the chest (such as infections, inflammation, cancer); nervous-system disorders involving multiple nerves; abnormalities of muscles (known as “myopathy”); immune-mediated disorders; and possible hormonal deficiencies (such as inadequate production of thyroid hormone [known as “hypothyroidism”] or inadequate production of steroids by the adrenal gland [known as “hypoadrenocorticism” or “Addison’s disease”])
- Thyroid cancer—may put pressure on or actually invade the recurrent laryngeal nerves

Trauma

- Penetrating wounds (such as bite wounds) or blunt trauma to the neck
- Injury secondary to ingested foreign materials—bones; sticks; needles; pins

Cancer

- Primary cancer of the voice box (larynx) or spread of cancer into the tissues of the voice box (metastatic cancer)
- Dogs—a variety of cancers reported, including squamous cell carcinoma, rhabdomyosarcoma, undifferentiated carcinoma, oncocyoma, lipoma, thyroid carcinoma, mast-cell tumor, osteosarcoma, fibrosarcoma, and melanoma
- Cats—the predominant cancer is lymphoma; squamous cell carcinoma and adenocarcinoma also reported

RISK FACTORS

- Existing lung abnormalities (such as pneumonia, chronic airway disease, and/or fluid build-up in the space between the chest wall and the lungs [known as “pleural effusion”]) can have a significant impact on breathing and may increase breathing difficulties associated with diseases of the voice box or larynx



TREATMENT

HEALTH CARE

- Outpatient—while awaiting surgery, if stable
- Emergency—characterized by marked breathing distress; oxygen therapy combined with sedation and steroids (dexamethasone); active body cooling measures with intravenous fluids and ice; temporary surgical opening into the windpipe (trachea; procedure known as a “temporary tracheostomy”) may prove life-saving in the patient that is not responding appropriately to the emergency medical approach
- Avoid warm, poorly ventilated environments, as these further compromise normal cooling mechanisms and proper air exchange.
- Avoid use of collars or choke chains to minimize pressure on the voice box (larynx) or windpipe (trachea)

ACTIVITY

- Severe activity restriction for patients, pending surgery or when owner refuses surgery

SURGERY

- Paralysis—surgical management is the treatment of choice; variety of procedures reported but correction on one-side only is preferred; benefit of procedure depends on the surgeon’s experience and expertise
- Trauma—temporary surgical opening into the windpipe (temporary tracheostomy) may be life-saving and curative
- Cancer—surgical tumor removal may be curative; for squamous-cell adenocarcinoma, surgical removal, coupled with radiation therapy, is the management of choice; permanent surgical opening into the windpipe (permanent tracheostomy) may improve quality of life



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Acquired (condition that develops sometime later in life/after birth) paralysis of the voice box or larynx in dogs, when surgery is declined—may benefit from mild sedatives (such as acepromazine, promazine, or diazepam) and steroids (prednisone)
- Lymphoma (cats)—potentially responsive to chemotherapy



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor for aspiration pneumonia
- Improvement in activity and exercise tolerance—reported by owners after effective surgery

PREVENTIONS AND AVOIDANCE

- Affected dogs of breeds in which hereditary transmission of paralysis of the voice box or larynx has been documented, should not be used for breeding purposes

POSSIBLE COMPLICATIONS

- Recurrence of clinical signs—with tumor regrowth; with inadequate surgery to treat paralysis
- Development of scar tissue that blocks the voice box or larynx (known as “laryngeal web formation”) has been seen in dogs after surgically removing both vocal cords; follow-up surgery and treatment with steroids may be necessary
- Increased risk of aspiration pneumonia—after any surgical procedure involving the voice box or larynx, as surgery places the larynx in a “fixed-open position,” eliminating its protective function during swallowing or regurgitation
- Risk of aspiration—particularly high if evidence of aspiration noted before surgical treatment of paralysis, and when swallowing disorders are present as well

EXPECTED COURSE AND PROGNOSIS

- Paralysis—long-term prognosis good to excellent with successful surgery; with unsatisfactory initial surgery, additional surgery may improve prognosis
- Trauma—progress usually satisfactory with conservative management, even after emergency tracheostomy
- Cancer—squamous-cell adenocarcinoma (dogs and cats): prognosis poor, even with radiation therapy; lymphoma (cats): prognosis depends on chemotherapy used and patient response



KEY POINTS

Paralysis

- Potential complications of heat exhaustion and impaired oxygenation, if surgery is not pursued
- Improved quality of life and normal life expectancy with successful surgery
- Potential genetic basis of paralysis of the voice box or larynx in certain dog breeds; affected dogs from these breeds should not be used for breeding purposes
- Increased risk for aspiration pneumonia after surgery

LEAD POISONING



BASICS

OVERVIEW

- Poisoning owing to sudden (acute) or long-term (chronic) exposure to some form of lead
- Blood lead levels are over 0.4 ppm

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs more commonly than cats

Mean Age and Range

- Mainly dogs less than 1 year of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Primarily gastrointestinal and nervous system signs
- Gastrointestinal signs often precede central nervous system signs; are most common with long-term (chronic), low-level exposure
- Central nervous system signs—occur more often with sudden (acute) exposure; more common in younger animals
- Ingestion of lead objects
- Vomiting
- Diarrhea
- Lack of appetite (known as “anorexia”)
- Abdominal pain
- Regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth) due to an enlarged esophagus (part of the digestive tract, the tube running from the throat to the stomach; condition known as “megaesophagus”)
- Sluggishness (lethargy)
- Hysteria
- Seizures
- Blindness
- Cats—sense of balance is altered (known as a “vestibular disorder”), with signs such as short, rapid movements of the eyeball (known as “nystagmus”) and wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”) reported

CAUSES

- Ingestion of some form of lead—paint and paint residues or dust from sanding; car batteries; linoleum; solder; plumbing materials and supplies; lubricating compounds; putty; tar paper; lead foil; golf balls; lead object (such as shot, fishing sinkers, drapery weights), leaded glass
- Use of improperly glazed ceramic food or water bowl
- Lead paint or lead-contaminated dust or soil are the most common source for exposure

RISK FACTORS

- Animal less than 1 year of age
- Living in economically depressed areas
- Living in old house or building that is being renovated



TREATMENT

HEALTH CARE

- Inpatient—first course of chelation, depending on severity of clinical signs; “chelation” is the use of specific chemicals to tie up the lead and allow it to be removed from the body

- Outpatient—chemicals administered by mouth to tie up the lead and allow it to be removed from the body (known as “oral chelators”)
- Balanced electrolyte fluids—Ringer’s solution; replacement of hydration deficit
- Flushing the stomach (known as “gastric lavage”) or whole bowel irrigation—may be indicated

SURGERY

- Surgical removal of lead objects from the gastrointestinal tract may be necessary



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Emptying or evacuation of gastrointestinal tract—saline cathartics; sodium or magnesium sulfate; “cathartics” are substances that evacuate the bowels
- Control of seizures—diazepam or phenobarbital
- Alleviation of fluid build-up in the brain (known as “cerebral edema”)—mannitol and dexamethasone
- Some evidence that antioxidants or thiol-containing drugs may be useful—examples include vitamins C and E, α -lipoic acid, N-acetylcysteine; optimal doses not determined
- B-vitamins, especially thiamine, also may be useful; optimal doses not determined
- Reduction of lead in the body (chelation therapy)— CaNa_2EDTA is a chelator for lead, patient may need multiple treatment courses, if blood lead concentration is high (allow 5-day rest period between treatment courses); succimer is a heavy metal chelator administered by mouth and is an alternative to CaNa_2EDTA (may administer per rectum, if clinical signs [such as vomiting] preclude oral administration)—advantages over other chelators: can be given by mouth, allowing for outpatient treatment



FOLLOW-UP CARE

PATIENT MONITORING

- Blood lead levels—should be less than 0.4 ppm; assess 10 to 14 days after stopping the use of specific chemicals to tie up the lead and allow it to be removed from the body (chelation therapy)

PREVENTIONS AND AVOIDANCE

- Test paint, dust, soil prior to animal access, if likelihood of lead contamination
- Determine source of lead and remove it from the patient’s environment

POSSIBLE COMPLICATIONS

- Permanent nervous system signs (such as blindness) occasionally

EXPECTED COURSE AND PROGNOSIS

- Signs should improve dramatically within 24 to 48 hours after initiating the use of specific chemicals to tie up the lead and allow it to be removed from the body (chelation therapy)
- Prognosis—favorable with treatment
- Uncontrolled seizures—guarded prognosis



KEY POINTS

- Potential of adverse human health effects of lead exposure; public health officials should be notified of lead poisoning in pets
- Determine the source of the lead

LEGG-CALVÉ-PERTHES DISEASE

(DEGENERATION OF THE “BALL” PORTION OF THE HIP JOINT)



BASICS

OVERVIEW

- Spontaneous degeneration of the femoral head and neck, leading to collapse of the hip joint (known as the “coxofemoral joint”) and osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage)
- The hip joint is composed of the “ball” (known as the “femoral head”) and the “socket” (known as the “acetabulum”); the “ball” sits on the “neck,” which is attached to the shaft of the femur, the long bone of the thigh

GENETICS

- Manchester terriers—inheritance pattern involving many factors, with a high degree of heritability
- Hereditary susceptibility likely

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Common among miniature-, toy-, and small-breed dogs
- Toy breeds and terriers—most susceptible
- Manchester terriers, miniature pinschers, toy poodles, Lakeland terriers, West Highland white terriers, and Cairn terriers—higher than expected incidence of disease as compared to other dog breeds

Mean Age and Range

- Most patients are 5 to 8 months of age
- Range—3 to 13 months of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Usually only one rear leg is involved; only 12% to 16% of cases are affected in both rear legs
- Lameness—usually gradual onset over 2 to 3 months; weightbearing; occasionally leg is carried (non-weightbearing)
- Pain on manipulation of the hip—most common
- Grating (known as “crepitation”) of the joint—inconsistent
- Decrease in size (known as “atrophy”) of the thigh muscles—nearly always noted

CAUSES

- Unknown
- Compression of the blood vessels serving the “ball” (femoral head) of the hip joint, with subsequent lack of blood flow, has been suggested as a cause, leading to the degeneration of the femoral head and neck and collapse of the hip joint

RISK FACTORS

- Miniature-, toy-, and small-breed dogs—increased risk
- Trauma to the hip region



TREATMENT

HEALTH CARE

- Rest and pain relievers (analgesics)—reportedly successful in alleviating lameness in a minority of patients
- Ehmer sling—successful in one patient; maintained for 10 weeks
- Subtle signs of onset often prevents early recognition and possibility of successful conservative treatment

- Surgical removal of the femoral head and neck (known as “femoral head and neck excision”) with early and vigorous exercise after surgery—treatment of choice

Postsurgery

- Physical therapy—extremely important for rehabilitating the affected limb
- Pain relievers (analgesics), anti-inflammatory drugs, and cold packing—for 3 to 5 days following surgery; important
- Range-of-motion exercises—extension and flexion; initiated immediately
- Small lead weights—attached as ankle bracelets above the hock joint; encourage early use of the treated limb

ACTIVITY

- Conservative therapy—restricted activity recommended
- Postsurgery—early activity encouraged to improve leg use

DIET

- Avoid obesity

SURGERY

- Surgical removal of the femoral head and neck (femoral head and neck excision)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs)—preoperative or postoperatively; minimize joint pain; reduce synovitis; NSAIDs include such drugs as carprofen, etodolac, meloxicam, deracoxib, firocoxib, buffered or enteric-coated aspirin—drugs should be administered only under the direction of your pet’s veterinarian
- Drugs intended to slow the progression of arthritic changes and protect joint cartilage (known as “chondroprotective drugs:” such as polysulfated glycosaminoglycans, glucosamine, and chondroitin sulfate)—little help in advanced disease; no evidence to suggest that these drugs prevent or reverse the disease process



FOLLOW-UP CARE

PATIENT MONITORING

- Conservative therapy—re-evaluate (physical examination, X-rays) to determine if surgery is needed
- Postsurgical progress checks—2-week intervals; necessary to ensure compliance with exercise recommendations

PREVENTIONS AND AVOIDANCE

- Discourage breeding of affected animals
- Do not repeat breedings that resulted in affected offspring

POSSIBLE COMPLICATIONS

- Limiting postoperative exercise may result in less than optimal limb function

EXPECTED COURSE AND PROGNOSIS

- Conservative therapy—reported to alleviate lameness after 2 to 3 months in about 25% of patients
- Surgical removal of the femoral head and neck (femoral head and neck excision)—good to excellent prognosis for full recovery (84%–100% success rate)



KEY POINTS

- Owners of Manchester terriers need to be aware of the genetic basis of the disease; discourage breeding affected dogs
- Recovery after surgical removal of the femoral head and neck (femoral head and neck excision) may take 3 to 6 months

LEPTOSPIROSIS



BASICS

OVERVIEW

- “Leptospirosis” is caused by disease-causing members of the bacterial genus *Leptospira*
- Sudden (acute) and long-term (chronic) diseases of dogs (mainly inflammation of the kidney [known as “nephritis”] and inflammation of the liver [known as “hepatitis”]) and other animals, including cats, although rarely
- Dogs—most disease caused by the serovars *Leptospira grippityphosa* and *Leptospira pomona*; “serovars” are subdivisions of a species that are different from other strains

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Rarely cats

Mean Age and Range

- Young dogs—more likely to exhibit severe disease
- Old dogs with adequate protection from vaccinations—seldom exhibit clinical disease, unless exposed to a serovar not in the vaccine

Predominant Sex

- Traditionally, male dogs more commonly affected; disputed by recent reports

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary with age and immune status of the animal, environmental factors that affect *Leptospira* survival, and disease-causing nature of the infecting serovar

- May have no clinical signs

Very Sudden (Peracute) Disease to Disease with Signs Over a Moderate Amount of Time (known as “Subacute Disease”)

- Fever
- Sore muscles
- Stiffness
- Shivering
- Weakness
- Lack of appetite (known as “anorexia”)
- Depression
- Vomiting
- Rapid dehydration
- Diarrhea—with or without blood
- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)
- Spontaneous cough
- Difficulty breathing (known as “dyspnea”)
- Increased thirst (known as “polydipsia”) and increased urination (known as “polyuria”) progressing to production of no urine (known as “anuria”)
- Bloody vaginal discharge
- Death—without clinical signs

Very Sudden (Peracute) to Sudden (Acute) Disease

- Rapid breathing (known as “tachypnea”)
- Rapid, irregular pulse
- Poor blood flow in the capillaries (smallest blood vessels; condition known as “poor capillary perfusion”)
- Vomiting blood (known as “hematemesis”)
- Passage of blood in the bowel movement or stool (known as “hematochezia”)
- Black tarry stools, due to the presence of digested blood (known as “melena”)

- Bleeding in the nose and nasal passages (known as “epistaxis” or a “nosebleed”)
- Widespread small, pinpoint areas of bleeding (known as “petechia”); bruises or purplish patches under the skin, due to bleeding (known as “ecchymoses”)
- Reluctance to move, overly sensitive to pain or touch (known as “hyperesthesia”) along the spine, stiff gait
- Inflammation of the moist tissues of the eyes (known as “conjunctivitis”)
- Inflammation of the nose (known as “rhinitis”)
- Blood in the urine (known as “hematuria”)
- Mildly enlarged lymph nodes (known as “lymphadenopathy”)

Long-Term (Chronic) Disease

- May have no apparent illness
- Fever of unknown origin
- Increased thirst (polydipsia) and increased urination (polyuria)—long-term (chronic) kidney failure

CAUSES

- Dogs—*Leptospira canicola*, *Leptospira icterohaemorrhagiae*, *Leptospira pomona*, *Leptospira grippityphosa*, *Leptospira bratislava*, *Leptospira copenhagenii*, *Leptospira australis*, *Leptospira autumnalis*, *Leptospira ballum*, and *Leptospira bataviae*
- Cats—*Leptospira canicola*, *Leptospira grippityphosa*, *Leptospira pomona*, and *Leptospira bataviae*

RISK FACTORS

Transmission

- Direct—host-to-host contact via infected urine, postabortion discharge, infected fetus/ discharge, and sexual contact (semen)
- Indirect—exposure (via urine) to a contaminated environment (such as vegetation, soil, food, water, bedding) under conditions in which *Leptospira* can survive
- Disease agent—*Leptospira* serovar, each with its own disease-causing factors, infectious dose, and route of exposure

Host Factors

- Vaccine—protection is serovar-specific; prevents clinical disease as a result of specific serovar; may not prevent kidney colonization of *Leptospira* and subsequent shedding of the bacteria in the urine; serovars not included in the vaccine may infect and cause disease in vaccinated animal
- Outdoor animals or hunting dogs—exposure of moist tissues of the body (mucous membranes) to water; exposure of abraded or water-softened skin increases risk of infection

Environmental Factors

- Warm and moist environment; wet season (high rainfall areas) of temperate regions; low-lying areas (marshy, muddy, irrigated); warm humid climates of tropical and subtropical regions
- Environmental temperature range—7° to 10° C (44.6° to 50° F) to 34° to 36° C (93° to 96° F)
- Water—organism survives better in stagnant than in flowing water; neutral or slightly alkaline pH
- Organism survives 180 days in wet soil and longer in standing water
- Dense animal population—kennels and urban settings; increases chances of urine exposure
- Exposure to rodents and other wildlife



TREATMENT

HEALTH CARE

- Sudden (acute) severe disease—inpatient; extent of supportive therapy depends on severity of disease; kidney failure requires closely monitored, medically induced increased production of urine (known as “diuresis”)
- Dehydration and shock—intravenous fluids (such as lactated Ringer’s solution)
- Severe bleeding—blood transfusion may be needed in association with treatment for the blood-clotting disorder, known as “disseminated intravascular coagulopathy” or “DIC”
- Production of only small amounts of urine (known as “oliguria”) or no urine (known as “anuria”)—initially rehydrate; then give medications to increase production of urine (known as “diuretics”); peritoneal dialysis (a type of dialysis in which fluids are put into the abdomen and the lining of the abdomen [known as the “peritoneum”] acts as a filter to remove waste products from the blood; after a certain amount of time, the fluids and waste products are removed from the abdomen) may be necessary

ACTIVITY

- Suddenly (acutely) ill patients and patients with the presence of bacteria in their blood (known as “bacteremia”) or generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”)—restricted activity; cage rest; monitoring; and warmth

DIET

- Severely ill patients—often have lack of appetite (anorexia); provide nutrition through intravenous feeding for prolonged anorexia



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Procaine penicillin G—an antibiotic; administer until kidney function returns to normal
- Dihydrostreptomycin—an antibiotic; administer for 2 weeks to eliminate organism from kidney tissues; try streptomycin if no kidney failure (drug not available everywhere)
- Doxycycline—an antibiotic; administer for 2 weeks; use alone to clear *Leptospira* from the blood and urine
- Ampicillin or amoxicillin—antibiotics; may be used instead of penicillin; administer for 2 weeks
- Erythromycin—an antibiotic



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor blood work and urinalysis for kidney function and monitor blood work for liver function and electrolytes
- Monitor blood work (blood urea nitrogen [BUN] and serum creatinine) and urine specific gravity in dogs with kidney failure for indication of prognosis

PREVENTIONS AND AVOIDANCE

- Vaccine (dogs)—whole-cell bacterin vaccines contain the serovars *Leptospira canicola* and *Leptospira icterohaemorrhagiae*; promotes immunity to these serovars and protection from overt clinical disease; serovar specific; does not promote protection against other serovars present in nature; may not prevent colonization of the kidneys of *Leptospira*, resulting in a long-term (chronic) carrier state; a “carrier state” is one in which the animal has no signs of disease, but harbors *Leptospira* and can transmit it to other animals
- Newer subunit vaccine contains the serovars *Leptospira pomona*, *Leptospira icterohaemorrhagiae*, *Leptospira grippotyphosa*, and *Leptospira canicola*; claims are made that the vaccine provides protection from clinical disease and prevents kidney colonization of *Leptospira*
- Vaccines—vaccinate dogs per current label recommendations; bacteria-induced immunity lasts only 6 to 8 months and is serovar specific (no cross-protection outside of the serogroup); revaccination at least yearly; vaccinate dogs at risk (such as dogs that hunt, show dogs, and dogs with access to water/ponds) every 4 to 6 months, especially in areas where *Leptospira* is found (known as “endemic areas”); the veterinarian will assess the risk of exposure and will recommend a vaccination protocol for your pet
- Kennels—strict sanitation to avoid contact with infected urine; control rodents; monitor and remove carrier dogs until treated; isolate affected animals during treatment; “carrier dogs” are infected, but have no signs of disease—they harbor *Leptospira* and can transmit it to other animals
- Activity—limit access to marshy/muddy areas, ponds, low-lying areas with stagnant surface water, heavily irrigated pastures, and access to wildlife

POSSIBLE COMPLICATIONS

- Blood-clotting disorder (disseminated intravascular coagulopathy or DIC)
- Liver and/or kidney dysfunction may be permanent
- Inflammation of the iris and other areas in the front part of the eye (known as “uveitis”)
- Abortion

EXPECTED COURSE AND PROGNOSIS

- Most infections are subclinical or long-term (chronic); a “subclinical infection” is one in which the animal is infected, but has no signs of disease
- Prognosis guarded for sudden (acute) severe disease



KEY POINTS

- Leptospirosis has zoonotic potential from contaminated urine of affected dogs and their environment; “zoonotic diseases” can be passed from animals to people

SUDDEN (ACUTE) LIVER FAILURE



BASICS

OVERVIEW

- Sudden (acute) loss of more than 75% of functional liver tissue; occurs primarily because of sudden (acute), massive death of liver tissue (known as “hepatic necrosis”)
- The liver is the largest gland in the body; it has many functions, including production of bile (a fluid substance involved in digestion of fats); production of albumin (a protein in the plasma of the blood); and detoxification of drugs and other chemicals (such as ammonia) in the body

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- More common in dogs than in cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden (acute) onset
- Vomiting
- Small intestinal diarrhea—may be bloody
- Enlargement of the liver (known as “hepatomegaly”), with tenderness of the liver on feeling the abdomen
- Bleeding
- Yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)
- Brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”)
- Seizures

CAUSES

Drugs

- Many drugs have been reported to cause sudden (acute) liver failure (such as azole antifungal drugs, azathioprine, nonsteroidal anti-inflammatory drugs [NSAIDs], acetaminophen, diazepam [cats], steroids, methimazole [cats], phenobarbital [dogs], sulfa drugs [dogs], and tetracycline)
- Any drug may be associated with sudden (acute) liver failure

Biological Toxins

- *Amanita* mushrooms, aflatoxins, blue-green algae

Toxins

- Heavy metals (such as lead, zinc, copper)
- Phenols (especially cats)

Infectious Agents and Bacterial Toxins (known as “Endotoxins”)

- Intestinal bacteria—*Clostridium perfringens*; *Clostridium difficile*; gram-negative bacteria
- Food poisoning—*Staphylococcus*; *E. coli*; *Salmonella*

Thermal Injury

- Heatstroke
- Whole-body increased body temperature (known as “hyperthermia”) treatments for cancer

Low Levels of Oxygen in the Liver (known as “Hepatic Hypoxia”)

- Blood clots (known as “thromboembolic disease”)
- Shock
- Blood-clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”)
- Sudden (acute) circulatory failure, from any cause

RISK FACTORS

- Administration of any potentially liver-toxic substance or drug

- Exposure to environmental toxins (such as *Amanita* mushroom, food-borne aflatoxins)
- Indiscriminate ingestion of substances that are potentially liver toxic



TREATMENT

HEALTH CARE

- Inpatient—intensive care required
- Control potential bleeding/clotting disorders with vitamin K₁, fresh frozen plasma, or fresh whole blood
- Fluids
- Colloid replacement—colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume; plasma preferred; hetastarch next best alternative
- Potassium and glucose—supplement as necessary; may decrease severity of signs of hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)
- Phosphate—supplement judiciously; low phosphate may aggravate hepatic encephalopathy
- Supplement oxygen, as needed

ACTIVITY

- Restricted activity promotes healing and regeneration of the liver

DIET

- Vomiting—withhold food and water by mouth (so called “NPO”) until controlled; use medications to control vomiting (known as “antiemetics”)
- Nutrition via the gastrointestinal tract (as with a feeding tube)—small volume, frequent meals to optimize digestion and absorption of nutrients and to minimize formation of intestinal toxins that may contribute to hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)
- Nutrition through intravenous route (known as “parenteral nutrition”)—may be used as a partial means of providing nutrition (recommended for short-term lack of appetite) to minimize breakdown of muscle (catabolism) or as a total means of providing nutrition (known as “total parenteral nutrition” or “TPN”), which is recommended if the pet has lack of appetite of more than 5 days and providing nutrition via the gastrointestinal tract is not possible
- Diet composition—use normal protein (nitrogen) content, if pet is tolerant; moderate protein restriction in pets with hepatic encephalopathy; strive to maintain a positive nitrogen balance that is essential for liver regeneration
- Supplemental vitamins are essential—water-soluble vitamins (vitamin B complex, vitamin C); vitamin K₁; vitamin E



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Drugs to Control Vomiting (known as “Antiemetics”)

- Metoclopramide—for mild or infrequent vomiting
- Ondansetron
- Chlorpromazine—for severe vomiting; may be administered by injection under the skin (SC or subcutaneous administration) or into muscle (IM) or administered rectally
- Histamine H₂-blocker—famotidine, if intestinal bleeding is present

Drugs for Hepatic Encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)

- Lactulose
- Metronidazole

Drugs for Fluid Build-Up in the Brain (Cerebral Edema) Associated with Hepatic Encephalopathy

- Mannitol
- Furosemide—medication to remove excess fluid from the body (known as a “diuretic”); increases removal of fluid and reduces production of cerebrospinal fluid; monitor hydration and serum potassium to avoid dehydration and low levels of potassium in the blood

(known as “hypokalemia”), which may worsen hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)

Drugs for Blood-Clotting Disorder (known as “Coagulopathy”)

- Fresh whole blood or fresh frozen plasma—to provide substances necessary for clotting in cases with clinically significant bleeding

Free Radical Scavengers and Antioxidants

- For ongoing liver-cell damage, reperfusion injury, and low levels of oxygen in the tissues (known as “hypoxia”)
- Vitamin E
- Vitamin C
- N-acetylcysteine—primarily used in the treatment of acetaminophen toxicity
- S-adenosylmethionine (SAME, Denosyl®-SD4); has several effects, including the promotion of liver-cell regeneration

Liver Protectants

- Silibinin (milk thistle), effectiveness reported for treatment of *Amanita* mushroom toxicity and certain other toxins
- Ursodeoxycholic acid—if long-term (chronic) liver injury or very high bile acids persist; bile acids are produced by the liver and are involved in fat digestion



FOLLOW-UP CARE

PATIENT MONITORING

- Temperature, pulse, respiration, and mental status
- High vigilance for infection, especially hospital-related infection (such as caused by use of catheters)
- Body weight—twice daily to guide fluid therapy; body weight and body condition scoring (estimate of weight status [under or overweight] as compared to normal weight) weekly to appraise nitrogen and energy balance
- Serum biochemical tests (acid–base, electrolyte balances [especially potassium and phosphate], and glucose)
- Blood tests specifically related to the liver (liver enzyme activities and bilirubin concentration)—every 2 to 3 days, until improvement

PREVENTIONS AND AVOIDANCE

- Vaccinate dogs against infectious canine hepatitis virus
- Avoid indiscriminate ingestion of drugs or toxins associated with liver toxicity
- Remove potential toxins from environment
- Consider long-term (chronic) use of medications that are potentially liver toxic; discuss use of medications and possible side effects with your pet’s veterinarian

POSSIBLE COMPLICATIONS

- Low blood sugar (hypoglycemia)
- Blood-clotting disorder (disseminated intravascular coagulopathy or DIC)
- Uncontrolled gastrointestinal bleeding
- Brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (hepatic encephalopathy)
- Long-term (chronic) liver insufficiency, progressive damage and scarring of the liver (known as “cirrhosis”)
- Sudden (acute) kidney failure
- Death

EXPECTED COURSE AND PROGNOSIS

- Prognosis—depends on extent of liver tissue destroyed and effectiveness of supportive care



KEY POINTS

- Sudden (acute) liver failure is a serious condition; some patients die even with optimal treatment
- An underlying cause for the death of the liver tissue (necrosis), such as exposure to a drug or toxin, should be investigated; however, it often will not be confirmed

BACTERIAL INFECTION OF THE LOWER URINARY TRACT



BASICS

OVERVIEW

- Result of bacterial colonization of the urinary bladder and/or upper portion of the urethra (the tube from the bladder to the outside, through which urine flows out of the body); the lower urinary tract includes the urinary bladder and the urethra

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- More common in dogs than in cats

Mean Age and Range

- All ages affected, but occurrence increases with age because of a greater frequency of other urinary tract problems (such as urinary stones [known as “uroliths”], prostate disease, and tumors) that increase the likelihood of secondary bacterial infection of the urinary tract
- Uncommon in cats 6 years old or younger; common in cats 10 years old or older

Predominant Sex

- More common in female than in male dogs
- Occurrence in male and female cats is similar

SIGNS/OBSERVED CHANGES in the ANIMAL

- None in some patients
- Frequent voiding of small volumes of urine (known as “pollakiuria”)
- Difficulty or painful urination (known as “dysuria”)
- Urgency to urinate (or an apparent loss of ability to control urination during periods of confinement)
- Urinating in places that are not customary (such as in the house)
- Bloody urine (known as “hematuria”) and cloudy or malodorous urine in some patients
- Sudden (acute) infection—bladder or urethra may seem tender on palpation during physical examination
- Palpation of the bladder may stimulate urination
- Long-term (chronic) infection—wall of the bladder or urethra may be thickened or abnormally firm
- Secondary infection—signs related to the underlying problem

CAUSES

- Most common bacteria that cause lower urinary tract infection—*Escherichia*, *Staphylococcus*, and *Proteus* (more than half of all cases)
- Common bacteria that cause lower urinary tract infection—*Streptococcus*, *Klebsiella*, *Enterobacter*, *Pseudomonas*, and *Corynebacterium*

RISK FACTORS

- Conditions that cause the urine to remain in the bladder for prolonged time periods (known as “urine stasis”) or incomplete emptying of the bladder
- Conditions that disrupt normal lower urinary tract lining defenses (those properties that protect the lower urinary tract from bacterial colonization)
- Conditions that reduce or bypass anatomic and functional barriers to bacteria moving up the urinary tract (such as loss of muscle tone or length of the urethra)
- Conditions that compromise the antibacterial properties of urine itself (such as changes in urine pH or concentration and low levels of urea and certain organic acids)



TREATMENT

HEALTH CARE

- Treat as outpatient, unless another urinary abnormality (such as urinary tract blockage or obstruction) requires inpatient treatment

ACTIVITY

- Unrestricted
- Regulating urination to coordinate with antibacterial drug treatments may improve therapeutic efficacy

DIET

- Restrictions not necessary, but may be indicated for other urinary tract diseases (such as kidney failure or urinary tract stones [urolithiasis])

SURGERY

- Except when a another urinary tract disorder requires surgical intervention, management of bacterial infection of the lower urinary tract does not involve surgery



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics—suggested antibiotics include penicillin for *Staphylococcus*, *Streptococcus*, or *Proteus*; trimethoprim-sulfadiazine for *Escherichia coli*; cephalexin for *Klebsiella*; tetracycline for *Pseudomonas*
- Bacterial culture and sensitivity testing provides identification of bacteria present in urine and information about antibiotics to which the bacteria is susceptible; if the infection does not respond as expected to the first antibiotic prescribed, base choice of antibiotics on results of sensitivity test
- Antibacterial drugs usually are most effective when given every 8 hours; however, fluoroquinolones and trimethoprim-sulfa products are effective when given every 12 hours—give antibiotics and any other medications as directed by your pet's veterinarian
- For sudden (acute), uncomplicated bacterial infection of the lower urinary tract—treat with antimicrobial drugs for 7 to 10 days; long-term (chronic) bacterial infection of the bladder (known as “cystitis”) may need treatment for up to 4 to 6 weeks; duration of treatment for complicated lower urinary tract infection depends on the underlying problem
- Low-dose, bedtime antibacterial therapy can be used to prevent infections in animals that have frequent recurrence of bacterial infections; the drug should be given after the animal has urinated for the last time each evening
- Other possible antibiotics include enrofloxacin and nitrofurantoin, as well as ceftiofur, gentamicin, or amikacin, which must be given by injection



FOLLOW-UP CARE

PATIENT MONITORING

- When the beneficial effects of the antibiotic treatment is in doubt, culture the urine 2 to 3 days after starting treatment—if the drug is effective, the bacterial culture will be negative
- Continue treating at least 1 week after resolution of blood in the urine (hematuria); white-blood cells or pus in the urine (known as “pyuria”); and protein in the urine (known as “proteinuria”)—failure of urinalysis findings to return to normal while an episode of urinary tract infection is being treated with an effective antibiotic (that is, as indicated by negative urine culture) generally indicates some other urinary tract abnormality (such as urinary stones [uroliths], tumor)
- Rapid recurrence of signs when treatment is stopped generally indicates either another urinary tract abnormality or that the infection extends into some deep-seated site (such as the prostate or kidney)
- Successful cure of an episode of urinary tract infection is best demonstrated by performing a bacterial culture of the urine 7 to 10 days

after completing antibiotics

- Animals being given low-dose bedtime antibiotics for frequent re-infection should have a bacterial culture of the urine performed every 1 to 2 months

PREVENTIONS AND AVOIDANCE

- Avoid indiscriminate use of urinary catheters
- Animals with frequent re-infection can be given bedtime therapy to prevent re-infection

POSSIBLE COMPLICATIONS

- Associated health risks include development of urinary tract stones (urolithiasis) and extension of infection to other portions of the urinary tract (such as the kidneys) or beyond to the blood (known as “septicemia”), to the spine (known as “diskospondylitis”), and to the heart (known as “bacterial endocarditis”)
- Failure to detect or treat effectively may lead to bacterial infection/inflammation of the kidney (known as “pyelonephritis”) or formation of a particular type of urinary tract stone (known as “struvite uroliths”)

EXPECTED COURSE AND PROGNOSIS

- If not treated, expect infection to persist indefinitely
- Prognosis for animals with uncomplicated lower urinary tract infection is good-to-excellent
- Prognosis for animals with complicated infection is determined by the prognosis for the underlying abnormality



KEY POINTS

- Following recommendations for treatment and follow-up evaluations is crucial for optimum results

LUMBOSACRAL STENOSIS AND CAUDA EQUINA SYNDROME



BASICS

OVERVIEW

- Pressure to or damage of the nerves within the spinal canal in the area of the junction between the lumbar and sacral vertebrae; at this level of the spine, spinal nerves are located in the spinal canal (rather than spinal cord)—these spinal nerves within the spinal canal are known as the “cauda equina”
- Caused by narrowing of the lumbosacral spinal canal with compression of the seventh lumbar (L_7), sacral, or caudal nerve roots
- Syndrome refers to the clinical signs related to injury of these nerve roots
- The narrowing of the lumbosacral spinal canal can be a congenital condition, which is present at birth, or an acquired condition, which develops sometime later in life/after birth
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C_1 - C_7 ; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T_1 - T_{13} ; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L_1 - L_7 ; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae
- Each disk is composed of a central gel-like area, known as the “nucleus pulposus,” and an outer fibrous ring, known as the “annulus fibrosis”
- Degeneration of the intervertebral disks causes protrusion of disk material into the spinal canal; the protruded disk material causes pressure on the spinal cord
- Protrusion is defined as the disk bulging into the spinal canal with the fibrous ring of the disk being intact
- Two types of protrusion have been reported in dogs: sudden (acute) disk herniation (“slipped disk”) is Hansen type I and long-term (chronic) disk herniation is Hansen type II; Hansen type II involves degeneration of the disk, followed by bulging of the disk into the spinal cord with the fibrous ring remaining intact (protrusion)

GENETICS

- No known genetic basis

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Common in dogs
- Rare in cats

Breed Predispositions

- Congenital (present at birth)—small to medium dogs; border collies
- Acquired (condition that develops sometime later in life/after birth)—large-breed dogs; German shepherd dogs, boxers, rottweilers

Mean Age and Range

- Congenital (present at birth)—signs seen at 3 to 8 years of age
- Acquired (condition that develops sometime later in life/after birth)—average age at onset of signs is 6 to 7 years

Predominant Sex

- Congenital (present at birth)—none
- Acquired (condition that develops sometime later in life/after birth)—male

SIGNS/OBSERVED CHANGES in the ANIMAL

- Relate to varying degrees of compression of the seventh lumbar (L_7), sacral, and caudal nerve roots
- Lumbosacral pain—salient clinical feature; may be the only sign
- Sciatic nerve dysfunction—the sciatic nerve is the largest nerve in the body; it travels from the pelvis/hip down the thigh; dysfunction initially may cause lameness; may progress to rear leg weakness, muscle wasting, and abnormal reflexes
- Pudendal nerve-root involvement—the pudendal nerve starts from the sacral nerves and provides innervation to the area of the rectum, external genitalia, and perineum (the skin between the anus and external genitalia); pudendal nerve-root involvement may lead to inability to control urination (known as “urinary incontinence”) and/or inability to control bowel movements (known as “fecal incontinence”)
- Caudal nerve-root involvement—abnormal tail carriage; weakness to paralysis of the tail

- Both meninges (the membranes covering the spinal cord) and nerve-root compression—sensory disturbances that vary from unpleasant sensations to obvious low lumbar pain
- Extension of the rear legs or movement of the tail over the back reduces the lumbosacral spinal canal diameter and usually elicits a painful response
- Congenital (present at birth)—self-inflicted lesions secondary to pain are common

CAUSES

- Congenital (present at birth) malformation of the backbones (vertebrae), including transitional vertebra (abnormal development of the backbone at the junction between two vertebral types; in this case, at the junction of the lumbar and sacral vertebrae) or osteochondrosis of the sacral endplates; “osteochondrosis” is a disorder of bone formation in the growth plates (areas where bone grows in length in the young animal), in this case, involving the sacral backbones (vertebrae)
- Hansen type II disk protrusion
- Increase in size (known as “hypertrophy” or “hyperplasia”) of the inter-arcuate ligaments (ligaments located between adjacent backbones [vertebrae])
- Proliferation of the articular facets (surfaces of the backbone [vertebra] where it joins together with another back bone)
- Partial dislocation (known as “subluxation”) or instability of the area of the junction between the lumbar and sacral backbones (vertebrae)

RISK FACTORS

- Dogs, especially German shepherd dogs, with a lumbosacral transitional vertebra (abnormal development of the backbone at the junction between two vertebral types; in this case, at the junction of the lumbar and sacral vertebrae) have increased risk to develop the syndrome



TREATMENT

HEALTH CARE

- Lack of control of urination (urinary continence)—outpatient, pending surgery
- Lack of control of urination (urinary incontinence)—inpatient for initial medical management
- Lack of control of urination (urinary incontinence)—catheterize the bladder until adequate voluntary control of urination returns; monitor closely for urinary tract infection and administer appropriate antibiotics, if necessary

ACTIVITY

- After surgery—restrict for 4 weeks; then gradually return to athletic function
- Nonsurgical treatment—confinement and restricted leash walks, alone or combined with steroids, frequently alleviates pain; clinical signs often return with increasing levels of exercise

DIET

- Avoid obesity; excess weight increases stress on the spine

SURGERY

- Surgery to relieve pressure on the nerves (known as “decompression”)—preferred treatment; various surgical procedures may be performed



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs) or steroids—used to decrease inflammation; usually unsatisfactory
- Lack of control of urination (urinary incontinence)—administer appropriate antibiotics, if patient develops urinary tract infection



FOLLOW-UP CARE

PATIENT MONITORING

- Evaluate nervous system status
- Lack of control of urination (urinary incontinence)—monitor closely for urinary tract infection

POSSIBLE COMPLICATIONS

- Accumulation of fluid (serum) in the tissues, causing the development of a mass (known as “seroma formation”)—frequent sequela to surgery; can be managed effectively by cage rest and surgical drainage of the fluid
- Excessive scar formation in the surgical area—infrequent cause of recurrence of clinical signs
- Recurrence of signs more than 6 months after surgery

EXPECTED COURSE AND PROGNOSIS

- Vary with degree of nerve injury
- Low lumbar pain and mild nervous system deficits (dogs)—good prognosis after surgery; 70% to 80% have an excellent or good outcome
- Inability to control urination (urinary incontinence) or bowel movements (fecal incontinence) in dogs—guarded prognosis



KEY POINTS

- Without treatment, the pet will have progressive nervous system impairment of the rear legs, lack of control of urination (urinary incontinence) and bowel movements (fecal incontinence), and paralysis of the tail
- Rear-leg lameness and self-inflicted lesions result from pain associated with nerve-root irritation and compression
- Discuss surgical treatment with your pet’s veterinarian; the goal of surgery is to stop the progression of nervous system impairment and to remove the source of pain
- Some nervous system deficits may remain following surgery
- Medical management alone usually is unsatisfactory

LYME DISEASE



BASICS

OVERVIEW

- One of the most common tick-transmitted diseases in the world
- Caused by spirochete species of the *Borrelia burgdorferi* group (such as *B. burgdorferi*, *B. afzelii*, *B. garinii*)
- Dominant clinical feature (dogs)—recurrent lameness due to inflammation of the joints (known as “arthritis”); sometimes lack of appetite (known as “anorexia”) and depression; may develop kidney and rarely heart or nervous system disease
- Reported in horses, cattle, and cats
- Also known as “Lyme borreliosis” or “borreliosis”

GENETICS

- Genetic basis known for mice; suspected for people; not established for dogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and rarely cats

Breed Predilections

- Kidney disease: Labrador retriever, golden retriever, Bernese Mountain dog

Mean Age and Range

- Experimentally, young dogs (puppies) appear to be more susceptible to disease than do adult dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Recurrent lameness due to inflammation of the joints (arthritis)
- Sudden (acute) form lasts for only 3 to 4 days; recurs days to weeks later in the same or in other legs (known as “shifting-leg lameness,” characterized by lameness in one leg, then that leg appears to be normal and another leg is involved); one or more joints may be swollen and warm; a pain response is elicited by feeling the joint; responds well to antibiotic treatment
- Long-term (chronic) inflammation of several joints, in which the bones around the joints are not destroyed (known as “nonerosive polyarthritis”) is found in animals with prolonged infection without adequate treatment; may persist despite antibiotic therapy
- Affected dogs may walk stiffly with an arched back and may be sensitive to touch
- Fever, lack of appetite (anorexia) and depression may accompany inflammation of the joints (arthritis)
- Superficial lymph nodes close to the site of the infecting tick bite may be swollen
- Kidneys—reported glomerulonephritis with immune-complex deposition in the glomeruli leading to fatal kidney disease; “glomerulonephritis” is inflammation and accompanying dysfunction of glomeruli (plural of glomerulus) of the kidney; each kidney is composed of thousands of nephrons (the functional units of the kidney, each consisting of the glomerulus [a tuft of blood capillaries—the “blood filter”] and a series of tubes and ducts, through which the filtered fluid flows, as urine is produced); inflammation most commonly is due to the presence of immune complexes in the glomerulus
- Kidney failure (signs include vomiting; diarrhea; lack of appetite [anorexia]; weight loss; increased urination [known as “polyuria”] and increased thirst [known as “polydipsia”]; fluid build-up in the tissues, especially the legs and under the skin [known as “peripheral edema”] or fluid build-up in the abdomen [known as “ascites”])
- Heart abnormalities—reported, but rare; include complete heart block
- Nervous system complications—rare

CAUSES

- *Borrelia burgdorferi*—transmitted by slow-feeding, hard-shelled tick species of the genus *Ixodes* (such as *Ixodes scapularis* [the deer tick], *Ixodes ricinus*, *Ixodes persulcatus*)
- Infection—only after a tick (nymph or adult female) carrying *Borrelia* has been attached to the host for at least 18 hours

RISK FACTORS

- Roaming in tick-infested environment, where Lyme borreliosis is common (known as an “endemic area”)



TREATMENT

HEALTH CARE

- Outpatient
- Keep patient warm and dry

ACTIVITY

- Reduced activity advisable until clinical signs improve

DIET

- No change needed

SURGERY

- Tapping the joint and removing joint fluid (known as “aspiration of synovial fluid”) may be considered for diagnostic purposes



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Most commonly used antibiotics—doxycycline, amoxicillin, or azithromycin
- Antibiotics do not eliminate the infection; consequently, persistent infection with a very low bacterial burden remains; treatment significantly improves clinical signs and disease
- Recommended treatment period—4 weeks
- Steroids—initially may cause signs to improve; may cover up or mask effects of antibiotics for diagnostic purposes; may increase clinical signs later by decreasing the ability of the animal to develop a normal immune response (known as “immunosuppression”)
- Nonsteroidal pain medications—use judiciously to avoid covering up or masking signs; use only as directed by your pet’s veterinarian



FOLLOW-UP CARE

PATIENT MONITORING

- Improvement in sudden (acute) inflammation of the joints caused by *Borrelia* (known as “Lyme arthritis”) should be seen within 3 to 5 days of antibiotic treatment
- If no improvement within 3 to 5 days, consider a different diagnosis

PREVENTIONS AND AVOIDANCE

- Mechanical removal of ticks—groom animals daily; discuss appropriate technique for removing ticks from your pet with the veterinarian
- Prevention of tick attachment—products to kill ticks (known as “acaricides”) and tick repellents are available commercially as spot-on topical products, sprays or collars; any such product should be used only according to label directions (do not use permethrin on cats)
- Vaccines—are available commercially for dogs; talk to your pet’s veterinarian about the vaccine
- Tick population control in the environment—restricted to small areas; limited success by reducing deer and/or rodent population

POSSIBLE COMPLICATIONS

- Fatal kidney failure
- Heart block
- Central nervous system disorders

EXPECTED COURSE AND PROGNOSIS

- Recovery from sudden (acute) lameness expected 3 to 5 days after initiation of antibiotic treatment
- Disease may be recurrent with intervals of weeks to months; responds again to antibiotic treatment



KEY POINTS

- Treatment of Lyme disease requires regular administration of antibiotics as prescribed by your pet's veterinarian
- Prevent tick attachment—products to kill ticks (acaricides) and tick repellents are available commercially; any such product should be used only according to label directions (do not use permethrin on cats)

DILATION OF LYMPHATIC VESSELS IN THE GASTROINTESTINAL TRACT (LYMPHANGIECTASIA)



BASICS

OVERVIEW

- “Lymphatic vessels” are vascular channels (similar to veins) that transport lymph; “lymph” is a clear to slightly colored fluid that contains white-blood cells—it circulates through the lymphatic vessels removing bacteria and other materials from body tissues and it also transports fat from the small intestines; it eventually empties into the blood, returning tissue fluids into the general body circulation
- “Lymphangiectasia” is defined as the dilation of the lymphatic vessels in the gastrointestinal tract; the “gastrointestinal tract” includes the stomach, small intestines, and large intestines
- Lymphangiectasia is an obstructive disorder of the lymphatic system of the gastrointestinal tract, resulting in the loss of body proteins through the intestines (known as “protein-losing enteropathy”)

GENETICS

- A familial tendency for the condition in which proteins are lost from the body through the intestines (protein-losing enteropathy) has been reported for soft-coated wheaten terriers, basenjis, and Norwegian lundehunds; “familial” indicates a condition that runs in certain families or lines of dogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Increased likelihood of lymphangiectasia seen in soft-coated wheaten terriers, basenjis, Norwegian lundehunds, and Yorkshire terriers as compared to other dog breeds

Age

- Dogs of any age can be affected
- Most common in middle-aged dogs

Predominant Sex

- Increased likelihood of lymphangiectasia seen in female soft-coated wheaten terriers as compared to males
- No sex has been reported to be more likely to develop lymphangiectasia in other breeds

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs are variable
- Diarrhea—long-term (chronic), intermittent or continuous, watery to semisolid consistency; however, not all patients have diarrhea
- Build-up of fluid in the abdomen (known as “ascites”)
- Build-up of fluid under the skin (known as “subcutaneous edema”)
- Difficulty breathing (known as “dyspnea”) from build-up of fluid in the space between the chest wall and the lungs (known as “pleural effusion”)
- Weight loss
- Excessive gas formation in the stomach or intestines (known as “flatulence”)
- Vomiting

CAUSES

Primary or Congenital (present at birth) Lymphangiectasia

- Localized—intestinal lymphatic vessels only
- Diffuse lymphatic abnormalities (such as accumulation of milky fluid in the space between the chest wall and lungs [known as “chylothorax”]; swelling due to the accumulation of lymph caused by blockage of the lymphatic vessels and/or lymph nodes [known as “lymphedema”]; accumulation of milky fluid in the abdomen [known as “chyloabdomen”]; or blockage of the thoracic duct, through which lymph is emptied into the general circulation)

Secondary Lymphangiectasia

- Right-sided congestive heart failure; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

- Inflammation of the sac (known as the “pericardium”) around the heart, characterized by thickening of the sac (condition known as “constrictive pericarditis”)
- Budd-Chiari syndrome (condition in which blood flow is blocked in the veins of the liver)
- Cancer (lymphosarcoma)



TREATMENT

HEALTH CARE

- Mostly treated as outpatients
- May need hospitalization if complications due to low levels of albumin, a type of protein, in the blood (known as “hypoalbuminemia”) develop

ACTIVITY

- Normal

DIET

- Low-fat diet with high-quality protein
- Long-chain triglycerides stimulate intestinal lymph flow and may lead to increased intestinal protein loss
- Diets fortified with medium-chain triglycerides (MCTs) may be beneficial
- May feed medium-chain triglycerides (MCTs) to supplement fat and increase calorie intake
- Commercial sources of medium-chain triglycerides (MCTs)—MCT oil or Portagen® (Mead Johnson, Evansville, IN)
- Supplement with fat-soluble vitamins—A, D, E, and K
- Elemental diets also can be used; “elemental diets” are liquid diets that contain amino acids, carbohydrates, low levels of fats, vitamins, and minerals that can be absorbed without the need for digestion

SURGERY

- When intestinal lymphangiectasia is secondary to an identifiable lymphatic blockage or obstruction, consider surgery to relieve the obstruction
- Surgery to remove part of the sac (pericardium) around the heart (known as a “pericardiectomy”) may be indicated in cases of inflammation of the sac, characterized by thickening of the sac (constrictive pericarditis)
- Patients that benefit from surgical intervention are rare



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Try steroids, if dietary therapy alone is unsuccessful (steroid treatment is not intended to treat lymphangiectasia, but rather to treat coexistent inflammation of the stomach and/or intestines); prednisone can be administered, after remission of the disease, dosage slowly can be decreased to the lowest dose effective at controlling the disease
- If the patient is cobalamin (vitamin B12) deficient, cobalamin must be supplemented to achieve therapeutic response
- If secondary small intestinal bacterial overgrowth is suspected, the patient should be treated with antibiotics (tylosin); small intestinal bacterial overgrowth (“SIBO”) is a condition in which a high number of bacteria are found in the upper small intestine



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor body weight, serum protein concentration, and evidence of recurrent clinical signs (such as fluid build-up in the space between the lungs and chest wall [pleural effusion], in the abdomen [ascites], and/or under the skin [edema])

- Patients need to be re-evaluated dependent on severity of the disease process

POSSIBLE COMPLICATIONS

- Breathing difficulty from fluid build-up in the space between the lungs and chest wall (pleural effusion)
- Severe protein-calorie depletion
- Diarrhea that is resistant to medical treatment

EXPECTED COURSE AND PROGNOSIS

- Prognosis is guarded
- Some animals fail to respond to treatment
- Remissions of several months to more than 2 years can be achieved in some patients



KEY POINTS

- Unpredictable disease progression and response to treatment

LYMPHOMA—CATS



BASICS

OVERVIEW

- Lymphocytes are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes normally are involved in the immune process
- Lymphoma is cancer (malignancy) of lymphocytes that usually involves lymph nodes or other lymphatic tissue of the body
- Lymphoma in cats is found in various anatomic locations in the body, including the mediastinum (known as the “mediastinal form of lymphoma”)—the mediastinum is the center portion of the chest that contains the heart and other organs (except for the lungs); the gastrointestinal tract (known as the “alimentary form of lymphoma”); the kidneys (known as the “kidney or renal form of lymphoma”); multiple organs/tissues throughout the body (known as the “multicentric form of lymphoma”); and a single organ/tissue in the body (known as the “solitary form of lymphoma”)
- Another term for lymphoma is “lymphosarcoma”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Mean Age and Range

- Mean age of feline leukemia virus (FeLV)-positive cats with lymphoma—3 years
- Mean age of FeLV-negative cats with lymphoma—7 years
- Median age of cats with localized lymphoma, outside of the lymph nodes—13 years

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on anatomic form
- Mediastinal form (located in the center of the chest)—open-mouthed breathing; coughing; regurgitation; lack of appetite (anorexia); weight loss; the front part of the chest is very firm and resistant to gentle compression during physical examination
- Alimentary form (located in the gastrointestinal tract)—lack of appetite (anorexia); weight loss; sluggishness (lethargy); vomiting; constipation; diarrhea; black, tarry stools, due to the presence of digested blood (known as “melena”); frank blood in the stool; thickened intestines or abdominal masses
- Kidney or renal form—consistent with kidney failure (such as vomiting; lack of appetite [anorexia]; increased thirst [known as “polydipsia”]; increased urination [known as “polyuria”]; and sluggishness [lethargy]); large, irregular kidneys
- Multicentric form (located in multiple organs/tissues throughout the body)—possibly none in early stages; lack of appetite (anorexia), weight loss, and depression with progression of disease; enlargement of lymph nodes throughout the body
- Solitary form (located in a single organ/tissue)—depends on location; nasal lymphoma—usually sneezing, nasal discharge, and occasionally facial deformity; spinal-cord lymphoma—quickly progressing partial paralysis of the hindquarters may be seen; cutaneous (skin) lymphoma—itchiness (known as “pruritus”); bleeding (hemorrhage); or masses on the skin accompanied by hair loss (hair loss known as “alopecia”)
- All forms—fever; dehydration; depression; extreme weight loss with muscle wasting (known as “cachexia”) in some patients

CAUSES

- Feline leukemia virus (FeLV) infection—patients inconsistently test positive during illness (for example, 85% with the mediastinal form, 45% with the kidney form, 20% with the multicentric form, and 15% with the alimentary forms of lymphoma test positive on FeLV test)

RISK FACTORS

- Feline leukemia virus (FeLV) exposure



TREATMENT

HEALTH CARE

- Outpatient, whenever possible
- Radiation therapy—may be used for localized lymphoma; relapses outside the radiation field are not uncommon
- Consult a veterinary oncologist to help assess best option(s) for treatment

ACTIVITY

- Normal

DIET

- No change in most cases; may require dietary change if cat has kidney failure

SURGERY

- To relieve intestinal blockages or obstructions and to surgically remove individual tumors
- To obtain biopsy specimens for microscopic examination



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Chemotherapy—used in a combination or sequential protocol; some protocols have induction and maintenance periods
- Combination chemotherapy (induction)—vincristine, cyclophosphamide, cytosine arabinoside, and prednisone
- Combination chemotherapy (maintenance)—methotrexate, chlorambucil, prednisone, and vincristine
- Sequential chemotherapy—week 1: vincristine, l-asparaginase, and prednisone; week 2: cyclophosphamide; week 3: vincristine; week 4: methotrexate; then repeat the cycle; maintenance: lengthen the time between each treatment
- Other protocols (such as the University of Wisconsin-Madison protocol) can be used
- Recurrence of lymphoma—doxorubicin, vinblastine, actinomycin-D, mitoxantrone, nitrogen mustard, procarbazine, and lomustine
- Low-grade intestinal lymphoma has responded to Leukeran® and prednisone
- Prednisone alone—can be used to decrease clinical signs and improve how the cat feels; temporary response, not a cure



FOLLOW-UP CARE

PATIENT MONITORING

- Physical examination, complete blood count (CBC), and platelet count—before each weekly cycle of chemotherapy
- X-rays—as necessary

PREVENTIONS AND AVOIDANCE

- Avoid exposure to or breeding feline leukemia virus (FeLV)-positive cats

POSSIBLE COMPLICATIONS

- Low white-blood cell counts (known as “leukopenia”)
- Generalized bacterial infection (sepsis)

EXPECTED COURSE AND PROGNOSIS

- Depends on initial response to chemotherapy, anatomic type, feline leukemia virus (FeLV) status, and tumor burden
- Mean survival with complete remission—7 months
- Median survival with partial remission—2.5 months
- Median survival with no response to treatment—1.5 months
- Mediastinal form (located in the center of the chest)—about 10% of patients with live more than 2 years
- Median survival with alimentary form (located in the gastrointestinal tract)—8 months
- Median survival with peripheral multicentric form (located in multiple organs/tissues: “peripheral” refers to tissues away from the

center of the body)—23.5 months

- Median survival with kidney form—if feline leukemia virus (FeLV)-negative, 11.5 months; if FeLV-positive, 6.5 months
- Median survival with a low tumor burden—if feline leukemia virus (FeLV)-negative, 17.5 months; if FeLV-positive, 4 months
- Treated with prednisone alone—patients live 1.5 to 2 months
- Median duration of complete remission from localized lymphoma—114 weeks
- Can get long-term survival with low-grade lymphoma



KEY POINTS

- Cure is possible, but highly unlikely
- Goal is to induce remission and achieve a good quality of life for patients for as long as possible

LYMPHOMA—DOGS



BASICS

OVERVIEW

- A lymphocyte is a type of white-blood cell, formed in lymphatic tissue throughout the body; lymphocytes are further divided into T lymphocytes (which are involved in cell-mediated immunity; for example, graft rejection) and B lymphocytes (which produce antibodies as part of the immune process)
- Lymphoma is cancer (malignancy) of lymphocytes that usually involves lymph nodes or other lymphatic tissue of the body
- Proliferation of cancerous lymphocytes in solid tissues, primarily in lymph nodes, bone marrow, and visceral organs; T or B or non-T/non-B type lymphocytes may be involved
- Lymphoma in dogs is found in various anatomic locations in the body, including the mediastinum (known as the “mediastinal form of lymphoma”)—the mediastinum is the center portion of the chest that contains the heart and other organs (except for the lungs); the gastrointestinal tract (known as the “alimentary form of lymphoma”); the kidneys (known as the “kidney or renal form of lymphoma”); multiple organs/tissues throughout the body (known as the “multicentric form of lymphoma”); and a single organ/tissue in the body (known as the “solitary form of lymphoma”)
- Another term for lymphoma is “lymphosarcoma”

GENETICS

- The expression of the tumor suppressor gene p53 in this tumor type is rare
- Gain or loss of chromosomes is documented in cancerous lymphocytes in dogs
- Immunophenotypes (the genetic expression of cells in lymphoma) are significant in determining prognosis

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Reported high-risk breeds—boxer, basset hound, golden retriever, St. Bernard, Scottish terrier, Airedale terrier, and bulldog
- Reported low-risk breeds—dachshunds and Pomeranians

Mean Age and Range

- Usually 5 to 10 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on anatomic form and stage of disease
- All forms of lymphoma may have nonspecific signs, such as lack of appetite (anorexia); sluggishness (lethargy); weight loss
- Multicentric form (located in multiple organs/tissues throughout the body)—generalized, painless lymph-node enlargement (known as “lymphadenomegaly”) most common; may note distended abdomen, secondary to liver enlargement (known as “hepatomegaly”); spleen enlargement (known as “splenomegaly”); or fluid build-up in the abdomen (known as “ascites”)
- Alimentary form (located in the gastrointestinal tract)—vomiting; diarrhea; lack of appetite (anorexia); marked weight loss; abdominal discomfort; palpable abdominal mass; thickened gut loops; irregularities of the lining of the rectum
- Mediastinal form (located in the center of the chest)—coughing; difficulty swallowing; lack of appetite (anorexia); drooling; labored breathing; difficulty breathing (known as “dyspnea”); rapid breathing (known as “tachypnea”); exercise intolerance secondary to mass(es) and/or fluid build-up in the chest; muffled heart sounds due to fluid build-up between the chest wall and lungs (known as “pleural effusion”)
- Skin may have chronic and non-responsive plaque lesions; plaques may be raised, may fuse together, and may have discharge present
- Lymphoma involving tissues other than the lymph nodes (known as the “extranodal form”)—vary with the anatomic site; eyes—excessive sensitivity to light (known as “photophobia”), inflammation of the lining of the eyes (known as “conjunctivitis”), inflammation of the front portion of the eye, between the cornea and iris (condition known as “anterior uveitis”), bleeding in the back of the eye (known as “retinal hemorrhage”); central nervous system—seizures, dementia, paralysis; kidney—pain over the lumbar spine, kidney enlargement, kidney failure; heart—exercise intolerance or fainting (known as “syncope”), irregular heart beat

CAUSES

- No specific cause proven

RISK FACTORS

- Some breeds (such as boxer, Scottish terrier, golden retriever, German shepherd dog, poodle, basset hound) have higher than expected likelihood of this disease



TREATMENT

HEALTH CARE

- Chemotherapy can be very effective in prolonging good quality of life
- A veterinary oncologist should be consulted regarding treatment, whenever possible
- Some veterinary oncology centers combine chemotherapy with radiation therapy
- Inpatient—intravenous chemotherapy
- Outpatient—after remission, some protocols allow owner to administer drugs orally at home; owner should wear protective gloves when administering these drugs
- Radiation therapy—may be used to treat refractory lymph nodes (that is, those lymph nodes that do not respond to medical therapy), large mediastinal involvement, and solitary skin (cutaneous) areas
- Fluid therapy—may benefit patients with advanced disease; may benefit clinically ill, azotemic (in which excess levels of urea and other nitrogenous waste products are present in the blood; may be related to kidney failure and/or dehydration) and/or dehydrated patients
- Tapping the chest or abdomen to remove excessive fluid—recommended with marked fluid build-up between the chest wall and lungs (pleural effusion) or fluid build-up in the abdomen (abdominal effusion)

ACTIVITY

- Restrict in patients with low white-blood cell (WBC) count or platelet count

DIET

- As advised by your veterinarian

SURGERY

- Usually indicated for biopsy only
- May be very helpful in specific circumstances, such as bowel blockage or obstruction, if the tumor is not responsive to chemotherapy
- Rarely successful in treating lymphoma, unless cancer is limited to one accessible site



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- As advised by your veterinarian and veterinary oncologist
- Combination chemotherapy—many protocols exist and some have superior remission and survival times, but toxicity may be increased; various drugs may be used, such as vincristine, cyclophosphamide, prednisone, methotrexate, l-asparaginase, doxorubicin, chlorambucil; drug selection and order of use is based on particular protocol being followed
- Single-agent therapy (doxorubicin)—associated with remission and survival times that are similar to those for some combination chemotherapy
- Steroids alone—effective in the short term (1 to 2 months)
- Retinoids—may be used for skin (cutaneous) lymphoma; example is isotretinoin
- Many alternative treatment protocols exist
- Lomustine (CCNU) or dacarbazine (DTIC)—may use for cases that do not respond well to other chemotherapeutic protocols
- Some centers combine chemotherapy with radiation therapy



FOLLOW-UP CARE

PATIENT MONITORING

- As advised by your veterinarian and veterinary oncologist
- Physical examination and microscopic evaluation of cells or tissue—all lymph nodes that do not respond to treatment
- Complete blood count (CBC) and platelet count
- After 2 to 3 courses of chemotherapy treatments, repeat tests with previously identified abnormal results before administering next treatment to confirm response
- Echocardiography and electrocardiography (ECG)—periodically during and after doxorubicin administration to identify development of drug-related heart toxicity

POSSIBLE COMPLICATIONS

- Low white-blood cell count (known as “leukopenia”) and low neutrophil count (neutrophils are a specific type of white-blood cell that fight infection; low neutrophil count is known as “neutropenia”)
- Vomiting and diarrhea
- Lack of appetite (anorexia)
- Heart toxicity—potential side effect of doxorubicin
- Hair loss (known as “alopecia”)
- Inflammation of the pancreas (known as “pancreatitis”)
- Presence of pus-forming bacteria and their poisons in the blood or tissues (known as “sepsis”)
- Tissue sloughing—chemotherapeutic drugs tend to be very caustic; if the drug leaks into the tissues as it is being administered intravenously, tissue damage may result and the tissues may slough (shed or fall off)

EXPECTED COURSE AND PROGNOSIS

- Immunophenotypes (the genetic expression of cells in lymphoma) are significant in determining prognosis
- Survival depends on the type of lymphoma, location, response to treatment, and aggressiveness of the treatment
- Median duration of first remission with combination chemotherapy or doxorubicin—6 to 12 months; 58% to 90% of patients achieve complete remission
- Median survival time with combination chemotherapy is often 9 to 12 months or more
- Mediastinal form (located in the center of the chest) of lymphoma and/or high levels of calcium in the blood (known as “hypercalcemia,” which can be seen in cases of lymphoma)—poorer prognosis
- Primary central nervous system lymphoma, diffuse gastrointestinal lymphoma, and multiple skin lesions due to lymphoma (cutaneous forms)—associated with poor response to treatment



KEY POINTS

- Usually a fatal disease, but many patients can live a long time in remission and enjoy a good quality of life during treatment
- Chemotherapy is rarely curative, and relapse usually occurs
- Side effects of chemotherapy drugs depend on the type used, but usually are associated with the gastrointestinal tract and bone marrow
- Most dogs have low white-blood cell counts (leukopenia) by day 7 to 10 of chemotherapy
- Response rate of 70% to 80% is seen with most chemotherapy protocols
- Quality of life is good while the patient is receiving chemotherapy and while it is in remission

MALOCCLUSION OF TEETH IN DOGS



BASICS

OVERVIEW

- “Occlusion” is the relationship or contact between the biting (known as “incising”) and chewing (known as “masticatory”) surfaces of the upper and lower teeth; “malocclusion” is any deviation in the relationship or contact between the biting and chewing surfaces of the upper and lower teeth
- Various types of malocclusion have been identified, including the jaws are of correct length, but specific teeth are positioned abnormally (known as “dental malocclusion”)—anterior crossbite; lance tooth; base-narrow canine teeth; and posterior crossbite; the lower jaw (mandible) is short in relation to upper jaw (maxilla) (a type of “skeletal malocclusion”)—tooth inclination or location may be improper (overshot); the lower jaw (mandible) is long in relation to upper jaw (maxilla) (another type of “skeletal malocclusion”)—tooth inclination or location may be improper (undershot); type of “wry bite” in which one quadrant of the jaw is elongated and one quadrant is shortened (another type of “skeletal malocclusion”)
- Accurate assessment of abnormalities of occlusion will help determine if treatment is warranted and what treatment is appropriate
- Deciduous teeth are the “baby teeth” that are the first set of teeth to erupt in the immature animal; these teeth normally “fall out” when the permanent teeth erupt
- Permanent teeth are the second set of teeth to erupt and are the teeth of the adult animal

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Breed predilection for certain malocclusions (such as lance teeth in Shetland sheepdogs)

Mean Age and Range

- No age predilection, though malocclusion usually is apparent after eruption of teeth (deciduous or permanent)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary greatly according to type, extent, and consequent injuries caused by the malocclusion
- May be associated with open or closed bites or overcrowding of the teeth
- Disease of the gums and supporting tissues of the teeth (known as “periodontal disease”)—may result from crowding or misalignment of teeth
- Soft-tissue defects—from traumatic tooth contact; may be seen in the floor of the mouth and palate; palatal trauma may eventually result in oronasal fistula formation; an oronasal fistula is an abnormal opening between the mouth and the nose
- Fractures or abnormal wear (attrition) of teeth—may result from improper tooth contact

CAUSES

- Congenital (present at birth) or hereditary factors—skeletal malocclusions and breed predilection
- Impediment to tooth eruption—operculum (the flap of tissue covering the unerupted tooth); retention of soft-tissue covering
- Delayed eruption of deciduous or permanent teeth
- Retention or delayed loss of deciduous teeth
- Traumatic injury affecting the jaws or teeth



TREATMENT

HEALTH CARE

- Not every malocclusion needs correction
- If the bite is functional and nontraumatic to the animal, treatment may not be necessary
- Extraction of the tooth or decreasing the height of the crown of the tooth (known as “crown reduction”) with pulp capping of offending teeth often can be effective treatment

- Orthodontic treatment usually is based on prevention of improper contact trauma, wear, or injury to hard or soft tissues

DIET

- Prevent chewing of items and provide a soft diet until the orthodontic appliance is removed, if orthodontic treatment is used

SURGERY

- Careful and gentle extraction of the maloccluded deciduous tooth to remove inappropriate physical impediment (known as “interceptive orthodontics”) in hopes that the permanent tooth will erupt in the appropriate position; when performed at least 4 weeks prior to permanent tooth eruption, success rate greater than 80% is not uncommon
- Careful and gentle extraction of the maloccluded deciduous tooth in hopes that the short jaw will be released from the bite interlock, allowing it to grow (if the genetic potential is present), prior to eruption of permanent teeth and reestablishment of bite interlock; performed at least 6 weeks prior to permanent tooth eruption, success rate less than 20% is common



FOLLOW-UP CARE

- Examine the orthodontic appliance twice daily, if orthodontic treatment is used
- Flush the mouth with an oral hygiene solution or gel, if orthodontic treatment is used
- Professional and dental home care to promote healthy gums and teeth, as directed by your pet’s veterinarian

PATIENT MONITORING

- For the orthodontically corrected occlusion to be stable, it needs to be self-retaining or it may tend to revert to malocclusion; examine at 2 weeks, 2 months, and 6 months after the treatment is complete to see if desired outcome is stable
- It is advisable at around 6 months following orthodontic therapy for X-rays to be taken and compared to pretreatment X-rays, to determine if all teeth still appear vital (alive) and to evaluate any root changes that may have occurred due to the pressures of tooth and root movement during orthodontics

PREVENTIONS AND AVOIDANCE

- Careful selection of puppies, with oral and general examination, as well as examination and history of sire and dame, prior to purchase
- Selective breeding, based on preferred breed characteristics
- Careful monitoring of deciduous and permanent tooth eruption for early detection and treatment, if required

POSSIBLE COMPLICATIONS

- Selective extraction of deciduous tooth prior to permanent tooth eruption—potential for injury to underlying permanent tooth buds, either by direct injury with extraction instruments or subsequent traumatic inflammation affecting tooth growth and maturity; injuries may result in tooth buds dying, teeth becoming nonvital (dead) as they erupt, roots developing improperly, lack of proper formation of the crown of the tooth or abnormal mineralization of the crown of the tooth
- Orthodontic movement of permanent teeth—several conditions involving abnormal roots or nonvitality of the tooth may result; these conditions are uncommon in properly managed orthodontic procedures

EXPECTED COURSE AND PROGNOSIS

- Course of treatment may vary with the type of malocclusion and the animal’s nature and habits (such as inappropriate chewing).
- Generally, most orthodontic cases take 1 to 7 months for movement and retention phase, depending on severity and if extrusion of tooth/teeth is required for stabilization of the bite; prognosis is good to excellent in most treated patients
- Prognosis is fair to good in most untreated malocclusions
- Complications in untreated cases—disease of the gums and supporting tissues of the teeth (periodontal disease); abnormal wear (attrition) or fractures of teeth; trauma to soft tissues; oronasal fistula (abnormal opening between the mouth and the nose) formation; drying or desiccation of exposed tooth surfaces, resulting in beige to brown discoloration
- Some cases DO NOT need or require orthodontic intervention; only routine observation for early detection and treatment of any secondary complications (such as periodontal disease, worn or chipped teeth) are advised



KEY POINTS

- “Occlusion” is the relationship or contact between the biting (known as “incising”) and chewing (known as “masticatory”) surfaces of the upper and lower teeth; “malocclusion” is any deviation in the relationship or contact between the biting and chewing surfaces of the upper and lower teeth

- Accurate assessment of abnormalities of occlusion will help determine if treatment is warranted and what treatment appropriate
- Not every malocclusion needs correction
- Orthodontic treatment usually is based on prevention of improper contact trauma, wear, or injury to hard or soft tissues

MAMMARY GLAND TUMORS IN CATS

(BREAST TUMORS IN CATS)



BASICS

OVERVIEW

- Cancerous (malignant) and benign tumors of the breast (mammary glands) in cats

GENETICS

- Unknown
- Siamese and domestic shorthair—may have twice the risk of other breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats; breast (mammary gland) tumors are the third most common type of tumor seen in cats

Breed Predilections

- Siamese and domestic shorthair—higher reported incidence rates than other breeds

Mean Age and Range

- Mean—10 to 12 years of age
- Range—9 months to 23 years of age

Predominant Sex

- Spayed females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Firm, nodular mass, which may adhere to the skin, but not to underlying abdominal wall
- Approximately 60% of patients have multiple gland involvement; 33% have simultaneous involvement of both right and left mammary gland chains
- Any or all glands may be involved; slightly higher incidence observed for the two cranial glands (located near the front legs)
- Nipples—often red and swollen; may have tan or yellow fluid discharge
- Ulceration—noted in 25% of patients
- Infiltrated lymphatic vessels—may appear as subcutaneous (that is, under the skin), linear, beaded chains
- Rear legs—may have fluid build-up (known as “edema”) and be uncomfortable; temperature to rear legs may be abnormal owing to presence of tumor blocking circulation

CAUSES

- Unknown
- Strong association with prior use of progesterone-like drugs
- Increased estrogen and progesterone exposure, directly associated with age at spay (known as “ovariohysterectomy”)
- Cellular and genetic changes (including amplification of cyclin-A, p53 aberrations and reduced E-cadherin expression) have been implicated

RISK FACTORS

- Intact females have a sevenfold higher risk than do spayed females to develop breast tumors
- Genetic—Siamese
- Administration of progesterone-like drugs (such as megestrol acetate) to treat other conditions—associated with development of benign and cancerous masses
- Age at time of spay (ovariohysterectomy)—cats spayed prior to six months of age and cats spayed after six months, but prior to one year of age, had only 9% and 14%, respectively, of the risk of breast cancer (known as “mammary carcinoma”) development compared with intact cats



TREATMENT

HEALTH CARE

- Discharge after surgery, if stable
- Supportive fluids and appropriate antibiotics, as needed

SURGERY

- Radical mastectomy—patients without evidence of spread of the cancer (known as “metastasis”) on X-rays, regardless of tumor size; removal of all four glands of the affected chain significantly reduces the chance of local recurrence; include the lymph nodes (if large or if cancer involvement is suspected) on the same side of the body—lymph nodes are found under the front leg (“axillary lymph nodes”) and at the area between the rear leg and the body (“inguinal lymph nodes”)
- Tumors in both mammary chains—perform two radical mastectomies, timed usually 2 to 4 weeks apart
- Survival time may be increased with bilateral radical mastectomy
- Spay (ovariohysterectomy) may be performed at the same time as the mastectomy in intact female cats—may address ovarian and uterine disease



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Combination chemotherapy—doxorubicin and cyclophosphamide; repeat every 3 to 4 weeks; shown to induce short-term partial and complete responses in about half of patients with spread of the cancer (metastatic cancer) or with tumors that cannot be removed surgically (known as “nonresectable” disease)
- Other chemotherapeutic agents (carboplatin, mitoxantrone) may have activity
- Mitoxantrone—may substitute for doxorubicin
- No available biological response modifier has shown efficacy



FOLLOW-UP CARE

PATIENT MONITORING

- Complete physical examination—every two months; emphasis on checking previous incision line(s), remaining mammary glands, and axillary and inguinal lymph-node areas
- Chest X-rays—every 1 to 3 months

PREVENTIONS AND AVOIDANCE

- Spay (ovariohysterectomy)—cats had a 91% reduction in the risk of developing breast cancer (mammary carcinoma), if spayed prior to 6 months of age

POSSIBLE COMPLICATIONS

- Tumor—low red-blood cell count (known as “anemia”); reduced bone mass (known as “osteoporosis”); increased calcium levels in the blood (known as “hypercalcemia”); a blood-clotting disorder (known as disseminated intravascular coagulopathy” or “DIC”); fluid build-up in the abdomen (known as “ascites”); fluid build-up in the space between the lungs and chest wall (known as “pleural effusion”)
- Chemotherapy—heart condition characterized by flabby heart muscle (known as “dilated cardiomyopathy”); reduction of bone-marrow activity (known as “myelosuppression”), resulting in low numbers of red-blood cells, white-blood cells, and/or platelets; lack of appetite (known as “anorexia”); gastrointestinal toxicity; kidney insufficiency; liver disease (known as “hepatopathy”); chemotherapeutic agents themselves may be carcinogenic (capable of causing cancer) and mutagenic (capable of causing mutations in genes)

EXPECTED COURSE AND PROGNOSIS

- High incidence of recurrence (66% with conservative surgery) and spread of cancer (metastasis)
- Time to recurrence—related to the type of surgery; radical mastectomy disease-free interval, 575 days (survival, 800 days); conservative surgery disease-free interval, 325 days (survival, 500 days)
- Single most important prognostic factor—tumor size; median survival with tumor diameter greater than 3 cm, 6 months after surgery; median survival with diameter less than 2 cm, approximately 3 years
- Five doses of doxorubicin following surgery resulted in longer median disease-free interval (442 days) compared to cats that received fewer doses (104 days)
- HER-2 overexpression has been correlated with poor prognosis, similar to breast cancer in people



KEY POINTS

- Early detection and surgical removal of breast (mammary gland) tumors in cats is very important
- Many patients have advanced disease when first presented for examination by the veterinarian—average of 5 months after the tumors are first noticed
- Spay (ovariohysterectomy) at an early age (prior to 6 months) has a significant protective effect

MAMMARY GLAND TUMORS—DOGS



BASICS

OVERVIEW

- Benign tumors or cancer (malignant tumors) of the mammary glands in dogs
- “Mammary” refers to a breast or mammary gland
- The mammary glands produce milk to feed newborn puppies; they are located in two rows that extend from the chest to the inguinal area; the nipples indicate the location of the mammary glands

GENETICS

- Genetic basis is possible; some genes are identified frequently in cancer of the mammary glands
- Mutations of BRCA-1 reported in some dog mammary tumors

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilection

- Toy and miniature poodles, English springer spaniels, Brittanys, cocker spaniels, English setters, pointers, German shepherd dogs, Maltese, and Yorkshire terriers have been reported to have an increased risk of developing breast or mammary tumors compared to other breeds

Mean Age and Range

- Median age—about 10.5 years (range, 1 to 15 years of age)
- Uncommon in dogs less than 5 years of age

Predominant Sex

- Female; extremely rare in males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Usually slow-growing single or multiple masses in the mammary glands—about 50% of patients have multiple tumors
- May have superficial loss of tissue on the surface of the skin over the mammary tissue, frequently with inflammation (known as “ulceration”)
- May be freely movable—implies benign behavior
- May be fixed to skin or body wall—implies malignant behavior or cancer

CAUSES

- Unknown; likely hormonal

RISK FACTORS

- Circumstantial evidence incriminates hormone treatment with progestins and estrogen in combination, prolactin, and growth hormone



TREATMENT

HEALTH CARE

- Surgery—primary mode of treatment
- Chemotherapy—may be effective, but use of chemotherapy for breast or mammary tumors is reported infrequently

SURGERY

- Local surgical removal of a breast or mammary tumor (such as a simple, regional, or unilateral [one-sided] mastectomy) with wide and deep margins (at least 2 cm in all directions)—may be as effective in terms of disease-free interval as radical bilateral [both sides] mastectomy

- Spay or ovariectomy (OHE) in intact bitches at time of surgical removal of the breast or mammary tissue (mastectomy) may enhance survival



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Chemotherapy with doxorubicin—reported to have induced partial remission in two dogs for 12 and 16 months, respectively
- Chemotherapy with 5-fluorouracil (5-FU) plus cyclophosphamide following surgery is reported to increase survival over surgery alone
- Your dog's veterinarian may consult a veterinary oncologist (cancer specialist) for additional or updated information regarding chemotherapy



FOLLOW-UP CARE

PATIENT MONITORING

- As suggested by your dog's veterinarian and/or by a consulting veterinary oncologist (cancer specialist)
- Physical examination and chest X-rays—1, 3, 6, 9, and 12 months after treatment

PREVENTIONS AND AVOIDANCE

- Spayed before first heat or estrous cycle—0.5% risk of developing breast or mammary tumors compared to intact bitch; spaying before the first heat or estrus is suggested to markedly decrease the likelihood of developing mammary tumors
- Spayed before second heat or estrous cycle—8.0% risk of developing breast or mammary tumors compared to intact bitch
- Spayed after second heat or estrous cycle—26% risk of developing breast or mammary tumors compared to intact bitch
- Spayed after 2.5 years of age—no sparing effect on risk of developing breast or mammary tumors

POSSIBLE COMPLICATIONS

- Infection following surgery
- Splitting open or bursting along the incision line (known as “dehiscence”) following surgery
- Reduction of bone-marrow activity (known as “myelosuppression”), resulting in low number of red-blood cells, white-blood cells, and/or platelets, with chemotherapy
- Blood-clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”) with some types of breast or mammary tumors (especially inflammatory carcinomas)
- Distant spread of the cancer (known as “metastasis”) and death

EXPECTED COURSE AND PROGNOSIS

- Varies with type of breast or mammary tumor (for example, benign tumor or cancer), size and presence or absence of spread of cancer (metastasis)
- Surgery for tumors that have not spread may be curative
- Median survival after surgical removal of the breast or mammary tissue (mastectomy) with tubular adenocarcinoma—24.6 months
- Median survival after surgical removal of the breast or mammary tissue (mastectomy) with solid carcinoma—6.5 months
- Benign tumor—excellent prognosis after surgical removal of the breast or mammary tissue (mastectomy)
- Carcinoma less than 5 cm in diameter—usually a good prognosis, if excision is complete
- Regional lymph-node involvement, confirmed by microscopic evaluation, makes the prognosis worse



KEY POINTS

- Never watch a breast or mammary nodule to “see what happens”—a breast or mammary lump should never be left in place and

observed

- Always make a plan for evaluation and possible surgical removal of any lump in the mammary gland(s)
- Early detection and surgical intervention is best
- Spaying before the first heat or estrus markedly decreases the likelihood of developing breast or mammary tumors

MAST-CELL TUMORS



BASICS

OVERVIEW

- Tumor arising from mast cells
- Mast cells are connective tissue cells that contain very dark granules; the granules contain various chemicals, including histamine; they are involved in immune reactions and inflammation; mast cells can be found in various tissues throughout the body
- Mast-cell tumors are graded as well differentiated (Grade 1), intermediately differentiated (Grade 2), and poorly differentiated or undifferentiated (Grade 3); in general, the more differentiated the mast-cell tumor, the better the prognosis
- Differentiation is a determination of how much a particular tumor cell looks like a normal cell; the more differentiated, the more like the normal cell

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Dogs—boxers and Boston terriers
- Cats—Siamese, susceptible to histiocytic cutaneous mast-cell tumors

Mean Age and Range

- Dogs—mean age, 8 years
- Cats—mastocytic form occurs at mean age of 10 years
- Cats—histiocytic form occurs at mean age of 2.4 years
- Reported in animals less than 1 year of age and in cats as old as 18 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on the location and grade of the tumor

Dogs

- Tumor on the skin or under the skin (known as “subcutaneous”), may have been present for days to months
- Tumor may have appeared to fluctuate in size
- Recent rapid growth after months of inactive or subtle growth is common
- Recent onset of redness (known as “erythema”) and fluid build-up (known as “edema”) most common with high-grade skin and subcutaneous tumors
- Extremely variable; may resemble any other type of skin or subcutaneous tumor (benign and cancer [malignant]); may resemble an insect bite or allergic reaction
- Primarily a single skin or subcutaneous mass; but may have multiple masses located in various parts of the body (known as “multifocal” mast-cell tumors)
- Approximately 50% of mast-cell tumors are located on the trunk and perineum (area between the anus and vulva (female) or scrotum (male); 40% on extremities; 10% on the head and neck region
- Lymph nodes may be enlarged in or near the area of the tumor (known as “regional lymphadenopathy”)—may develop when a high-grade tumor spreads (metastasizes) to the lymph nodes
- Enlarged liver (known as “hepatomegaly”) and enlarged spleen (known as “splenomegaly”)—features of wide-spread (disseminated) mast-cell cancer

Cats

- Lack of appetite (known as “anorexia”)—most common complaint with mast-cell tumor of the spleen
- Vomiting—may occur secondary to mast-cell tumors of the spleen or gastrointestinal tract
- Skin mast-cell tumors—primarily found in the tissue under the skin (subcutaneous tissue); may be papular (small, solid elevations) or nodular, single or multiple, and hairy or without hair (known as “alopecic”) or have an ulcerated surface; slight predilection for the head and neck regions
- Mast-cell tumor of the spleen—enlarged spleen (splenomegaly) is only consistent finding
- Intestinal mast-cell tumor—firm, segmental thickenings of the small intestinal wall; spread (metastasis) to the mesenteric lymph nodes, spleen, liver, and (rarely) lungs

CAUSES

- Unknown

RISK FACTORS

- Hereditary
- Previous inflammation



TREATMENT

HEALTH CARE

Dogs

- Aggressive surgical removal of the mast-cell tumor and surrounding tissue—treatment of choice
- Microscopic evaluation of the entire surgically removed tissue—essential to determine completeness of surgical removal and predict the biologic behavior of the tumor; if tumor cells extend close to the surgical margins, perform a second aggressive surgery as soon as possible
- Lymph-node involvement, but no generalized involvement in other parts of the body—aggressive surgical removal of the affected lymph node(s) and the primary tumor required; follow-up chemotherapy useful to prevent further spread of tumor cells (metastasis)
- Primary tumor and/or affected lymph nodes cannot be excised for microscopic disease—chemotherapy may have short-term benefit to make the patient feel better (known as a “palliative benefit”) of 1 to 4 months
- Generalized spread of tumor cells (metastasis) to other parts of the body—surgical removal of primary tumor and affected lymph nodes are of minimal benefit, but chemotherapy may have short-term palliative benefit (less than 2 months)
- Radiation therapy—good treatment option for mast-cell tumor of the skin in a location that does not allow aggressive surgical removal; if possible, perform surgery before radiation therapy to reduce the tumor to a microscopic volume; tumors on an extremity respond better than do tumors located on the trunk

Cats

- Surgery—treatment of choice for mast-cell tumors of the skin
- Surgical removal of the spleen (known as “splenectomy”)—treatment of choice for mast-cell tumors of the spleen
- Surgical removal of the spleen (splenectomy) and chemotherapy—may be beneficial when mast cells are circulating in the blood (known as “mastocythemia”) accompanies mast-cell tumors of the spleen

SURGERY

- Excisional biopsy with wide margins reasonable for very small tumors
- Incisional biopsy of large mast-cell tumors is recommended to obtain a tumor grade, predict prognosis, and establish a treatment plan; consider pretreatment with antihistamine therapy prior to incisional biopsy
- Biopsy of lymph nodes and other suspicious internal organs—appropriate
- Complete surgical removal with 3-cm margins in all planes recommended for all moderate Grade 2, high Grade 2, and Grade 3 tumors; margins of 2 cm or less may be adequate for Grade 1 and low Grade 2 tumors
- Surgical removal of regional lymph nodes recommended for all high Grade 2 and Grade 3 tumors



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Combination chemotherapy— prednisone, vinblastine, cyclophosphamide; recent information suggests that lomustine may be more effective than cyclophosphamide
- Prednisone—short-term remission only when used alone (although occasional exceptions do occur); is beneficial in some cases to achieve reduction in tumor load before surgery
- Other chemotherapeutic drugs (such as lomustine, vinblastine, cyclophosphamide)—add to length of remission of prednisone-sensitive tumors
- Mast-cell tumor of the skin not controlled by surgery or radiation therapy—medical treatment appropriate; in author’s experience, prednisone and chemotherapy not beneficial for aggressive skin (cutaneous) tumors in cats

- Prednisone-resistant tumor—chemotherapy does not appear to be beneficial
- Intestinal tumor and systemic mastocytosis (abnormal proliferation of mast cells in various tissues throughout the body) after surgical removal of the spleen (splenectomy) in cats—prednisone and chemotherapy indicated
- Measurable tumor (dogs)—vincristine alone induced partial remission in 21% of patients
- Histamine-blocking agents (such as cimetidine)—helpful, particularly for systemic mastocytosis (abnormal proliferation of mast cells in various tissues throughout the body) or when massive histamine release is a concern



FOLLOW-UP CARE

PATIENT MONITORING

- Evaluate any new masses microscopically
- Evaluate regional lymph nodes at regular intervals to detect spread (metastasis) of Grade 2 to 3 tumors
- Check complete blood count (CBC) at regular intervals, if patient is receiving chemotherapy
- Check liver enzymes on serum biochemistry profile, if patient is on long-term lomustine therapy

POSSIBLE COMPLICATIONS

- Bleeding
- Bloody inflammation of the gastrointestinal tract (known as “hemorrhagic gastroenteritis”)
- Poor wound healing, if surgical margins inadequate

EXPECTED COURSE AND PROGNOSIS

Dogs

- Location of primary tumor is important prognostic factor: tumors located around the prepuce (known as “peripreputial”),; beneath the claw (known as “subungual”); around the anus (known as “perianal”); in the mouth (known as “oral”); and on the muzzle region are associated with more undifferentiated tumors and poorer prognosis
- Tumors of the inguinal, perineal and muzzle regions tend to be more aggressive than their histologic grade might suggest; these tumors should always be considered to have the potential for metastasis
- Historical survival data (Bostock) after surgery only indicates the following survival times—Grade 1, 77% alive; Grade 2, 45% alive; Grade 3, 13% alive; the relevance of these historical statistics is questionable as patients with mast-cell tumor currently undergo aggressive staging and are treated more aggressively with surgery (that is, surgical removal of regional lymph nodes)
- Lymph-node metastasis—degree of lymph-node involvement does affect prognosis; patients with Grade 2 mast-cell tumors with microscopic confirmation of lymph-node metastasis, without evidence of lymph-node enlargement, have a very good long-term prognosis when complete surgical resection of the primary tumor and lymph node is performed, followed by a 6-month chemotherapy regimen; survival time for patients with Grade 3 tumors also is improved, compared to less-aggressive surgery or no follow-up chemotherapy, but most do not survive beyond 1 year; when the lymph nodes are enlarged grossly, prognosis remains guarded (even when aggressive resection and chemotherapy are administered)
- Prednisone alone—effectively induced remission and prolonged survival time in 20% of patients with Grade 2 or 3 tumors; only one of the five responding patients had documented lymph-node metastasis when prednisone was initiated

Cats

- Single mast-cell tumor of the skin—prognosis excellent; rate of recurrence low (16% to 36%) despite incomplete excision; less than 20% of patients develop metastasis
- Survival after surgical removal of the spleen (splenectomy) for mast-cell tumor of the spleen—reports of greater than 1 year
- Concurrent development of mast cells circulating in the blood (mastocythemia)—prognosis poor; prednisone and chemotherapy may achieve short-term remission
- Intestinal tumor—prognosis poor; survival times rarely greater than 4 months after surgery



KEY POINTS

- Twenty percent of dogs diagnosed with a mast-cell tumor will have two or more unrelated mast-cell tumors in their lifetimes; each of these has the potential for being cured with appropriate surgical intervention
- Fine-needle aspiration and microscopic examination should be performed as soon as possible on any new mass

- Appropriate surgical excision should be done as soon as possible on any new mast-cell tumor

MEGACOLON



BASICS

OVERVIEW

- “Mega-” refers to large or oversized; “colon” is another term for the large intestine
- “Megacolon” is a condition of persistent, increased large-intestine diameter associated with long-term (chronic) constipation/obstipation and low-to-absent movement of the large intestines (known as “colonic motility”)
- “Constipation” is infrequent, incomplete, or difficult defecation with passage of hard or dry bowel movement (feces)
- “Obstipation” is constipation that is difficult to manage or does not respond to medical treatment, caused by prolonged retention of hard, dry bowel movement (feces); defecation is impossible in the patient with obstipation

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Dogs and cats—acquired (condition that develops sometime later in life/after birth) megacolon
- Cats—enlarged large intestine of unknown cause (so called “idiopathic megacolon”)

Breed Predispositions

- Some evidence for increased risk in Manx cats

Mean Age and Range

- Acquired (condition that develops sometime later in life/after birth) megacolon—none
- Idiopathic megacolon (enlarged large intestine of unknown cause)—middle-aged to old cats (mean age, 4.9 years; range, 1 to 15 years)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Acquired (condition that develops sometime later in life/after birth) megacolon—signs may be sudden (acute) or long-term (chronic)
- Idiopathic megacolon (enlarged large intestine of unknown cause)—typically a long-term (chronic) or recurrent problem; signs often present for months to years
- Constipation/obstipation (constipation that is difficult to manage or does not respond to medical treatment)
- Painful defecation or straining to defecate (known as “tenesmus”) with small or no fecal volume
- Hard, dry feces
- Infrequent defecation
- Small amount of diarrhea (often with mucus) may occur after prolonged painful defecation or straining to defecate (tenesmus)
- Occasional vomiting, lack of appetite (known as “anorexia”), and/or depression
- Weight loss
- Enlarged colon with hard bowel movement (feces) may be detected on physical examination
- Rectal examination may indicate an underlying (obstructive) cause and confirms the presence of hardened bowel movement in the large intestine (colon) or rectum (condition known as “fecal impaction”)
- Dehydration
- Scruffy, unkempt hair coat

CAUSES

- Idiopathic megacolon (enlarged large intestine of unknown cause)—cats
- Mechanical blockage or obstruction of the passage of bowel movement (feces)—pelvic fracture; foreign body or improper diet (especially bones); abnormal narrowing of the colon or rectum (known as a “stricture”); condition in which bowel movement (feces) becomes trapped and matted in the hair around the anus, blocking the anus (known as “pseudocoprostasis”); prostate disease; condition in which the muscles supporting the rectum weaken and separate, allowing the rectum and/or bladder to slide under the skin and causing swelling in the area of the anus (known as a “perineal hernia”); cancer; birth defect in which the anus or rectum does not have an opening (known as “anal atresia” or “rectal atresia,” respectively)
- Causes of difficulty defecating (dyschezia)—disease of the anus and/or rectum (such as inflammation of the anal sacs [known as “anal sacculitis”]; anal sac abscess; one or multiple draining tracts around the anus (known as “perianal fistulae”); inflammation of the lining of the rectum [known as “proctitis”]); trauma (fractured pelvis, fractured limb, dislocated hip, bite wound or laceration in the tissue around the anus, perineal [area between the anus and external genitalia] abscess)
- Metabolic disorders—low levels of potassium in the blood (known as “hypokalemia”), severe dehydration

- Various medications—examples include vincristine, barium, antacids, sucralfate, anticholinergics (used as preanesthetics or to treat diarrhea, such as atropine)
- Nervous system and/or muscular disease—congenital (present at birth) abnormalities of the spine (especially Manx cats); paralysis of the rear legs (known as “paraplegia”); spinal cord disease; intervertebral disk disease; abnormal function of the autonomic nervous system (known as “dysautonomia”); sacral nerve disease; sacral nerve trauma (such as a tail fracture/pull injury); trauma to nerves to the large intestine

RISK FACTORS

- Pain involving the rectum and/or anus and conditions (such as pelvic and limb fractures or diseases of the nerves and/or muscles) leading to inability to posture to defecate
- Prior pelvic fractures
- Possible association with low physical activity and obesity
- Perineal hernias; a “perineal hernia” develops when the muscles supporting the rectum weaken and separate, allowing the rectum and/or bladder to slide under the skin and causing swelling in the area of the anus



TREATMENT

HEALTH CARE

- Inpatient medical management; surgery may be indicated, if recurrent or severe problem
- Medical treatment—restore normal hydration, followed by anesthesia and manual evacuation of the colon using warm water enemas, water-soluble jelly, and gentle extraction of feces with a gloved finger or sponge forceps
- Continue long-term therapy at home
- Most patients require fluids to correct dehydration
- Continue fluid support until the patient is willing to eat and drink

ACTIVITY

- Encourage activity and exercise
- Restricted activity indicated in the postoperative period, if surgery is performed

DIET

- Many patients require a low-residue-producing diet; bulk-forming fiber diets can worsen or lead to recurrence
- A high-fiber diet is occasionally helpful
- A maintenance-type diet can be supplemented with products such as Metamucil® or pumpkin-pie filler

SURGERY

- An underlying blockage or obstructive cause requires surgical correction
- Avoid enema administration/colonic evacuation prior to surgical procedure to remove part of the colon (known as a “subtotal colectomy”)
- Surgical removal of a section of the colon, with connection of the ends of the remaining sections of the intestines (known as “ileorectal or colorectal resection and anastomosis”)—treatment of choice for idiopathic megacolon (enlarged large intestine of unknown cause) that does not respond to medical management
- Surgical removal of the colon (known as a “colectomy”) may be required with obstructive megacolon caused by irreversible changes in movement of the large intestines (colonic motility)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Can improve large intestinal movement (colonic motility) in less severe cases with cisapride, a gastrointestinal prokinetic drug; “gastrointestinal prokinetic drugs” are medications that improve the propulsion of contents through the stomach and intestines
- Stool softeners (such as lactulose) are recommended in conjunction with cisapride and diet

- Broad-spectrum antibiotics are recommended prior to emptying the colon and rectum of dry, hard bowel movement (feces) and during the time immediately surrounding surgery, if surgery is elected
- Docusate sodium can be used as a stool softener in place of lactulose



FOLLOW-UP CARE

PATIENT MONITORING

- Following surgical removal of part of the large intestine (colon) with connection of the ends of the remaining sections of the intestines (known as “colonic resection and anastomosis”)—for 3 to 5 days check for signs of splitting open or bursting along the incision line (known as “dehiscence”) and inflammation of the lining to the abdomen (known as “peritonitis”)
- Clinical deterioration warrants tapping the abdomen (known as “abdominocentesis”) and/or flushing the abdomen (known as “peritoneal lavage”) to detect leakage of intestinal contents through the incision site

PREVENTIONS AND AVOIDANCE

- Repair pelvic fractures that narrow the pelvic canal
- Avoid exposure to foreign bodies and feeding bones

POSSIBLE COMPLICATIONS

- Recurrence or persistence—most common
- Potential surgical complications include inflammation of the lining of the abdomen (peritonitis), persistent diarrhea, abnormal narrowing of the large intestine (stricture formation), and recurrence of obstipation (constipation that is difficult to manage or does not respond to medical treatment)
- Abnormal opening or hole in the large intestines (known as a “perforation”)

EXPECTED COURSE AND PROGNOSIS

- Historically, medical management has been unrewarding
- Cisapride appears to improve the prognosis with medical management in some patients, but may not suffice in severe or long-standing cases
- Postoperative diarrhea—expected; typically resolves within 6 weeks (80% of cats with idiopathic megacolon [enlarged large intestine of unknown cause] undergoing surgical removal of part of the colon [subtotal colectomy]), but can persist for several months
- Surgical removal of part of the colon (subtotal colectomy) is well tolerated by cats; constipation recurrence rates are typically low



KEY POINTS

- In idiopathic megacolon (enlarged large intestine of unknown cause) or with severe colonic injury, medical treatment often is lifelong and can be frustrating
- Recurrence is common
- Surgical removal of part of the colon (subtotal colectomy) is indicated, if medical treatment fails

MEGAESOPHAGUS

(ENLARGEMENT OF THE ESOPHAGUS)



BASICS

OVERVIEW

- The esophagus is part of the digestive tract; it is a muscular tube that runs from the throat to the stomach; food and liquids are moved through the esophagus by peristalsis (wave-like motion produced by contraction and relaxation of muscles) toward the stomach
- Megaesophagus is defined as a generalized, diffuse enlargement (dilation) of the esophagus, in which peristaltic movement is decreased to absent

GENETICS

- Congenital (present at birth)/inherited form: inherited in wire fox terriers as an autosomal recessive trait and in miniature schnauzers as an autosomal dominant or 60 % penetrance autosomal recessive trait
- Other breeds reported include dachshund, German shepherd dog, Great Dane, Irish setter, Labrador retriever, pug (one litter with 4 puppies affected), and Chinese shar pei
- Myasthenia gravis (a disorder of neuromuscular transmission characterized by muscular weakness and excessive fatigue) may be congenital in Jack Russell terriers, English springer spaniels, smooth fox terriers, and Samoyeds
- Acquired (present later in life/after birth) form: many diseases may have an association with megaesophagus, some have genetic susceptibilities

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats; dogs are affected more commonly than cats

Breed Predilections

- Dog: wire fox terrier, miniature schnauzer, dachshund, German shepherd dog, Great Dane, Irish setter, Labrador retriever, pug, Chinese shar pei
- Cat: Siamese and Siamese-related cats

Mean Age and Range

- Congenital (present at birth) cases present soon after birth or at weaning from liquid to solid foods
- Acquired (present later in life/after birth) cases may be seen at any age, depending on the cause of the megaesophagus

SIGNS/OBSERVED CHANGES in the ANIMAL

- Owners often report vomiting (forceful ejection of stomach contents up through the esophagus and mouth); the veterinarian must differentiate vomiting from regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth)
- Regurgitation (considered the hallmark sign of megaesophagus); difficulty swallowing (known as “dysphagia”); coughing and nasal discharge with aspiration pneumonia; ravenous appetite or lack of appetite; weight loss or poor growth; excessive drooling (known as “ptyalism”)
- Swelling of the neck, bad breath (known as “halitosis”); increased noises with breathing; nasal discharge; and fever (if aspiration pneumonia present); weight loss or extreme weight loss with muscle wasting (known as “cachexia”); weakness
- Other signs depend upon underlying cause of megaesophagus
- Animals should be assessed carefully for other muscle and/or nervous system deficits that may indicate generalized disease

CAUSES

Congenital (Present at Birth)

- Unknown cause (known as “idiopathic megaesophagus”)
- Myasthenia gravis (a disorder of neuromuscular transmission characterized by muscular weakness and excessive fatigue) is a rare cause

Acquired/Adult Onset

- Unknown cause (known as “idiopathic megaesophagus”) is most common type
- Neuromuscular disease: myasthenia gravis (25% of cases in dogs); systemic lupus erythematosus (an autoimmune disease that affects many organs and tissues of the body); inflammation of the muscles (known as “myositis”) or other muscle disease (known as “myopathic disease”); dysfunction of the autonomic nervous system (known as “dysautonomia;” more common in cats); distemper; inherited diseases in which normal glycogen (the body’s carbohydrate reserve) metabolism is altered (known as “glycogen-storage disease”); tetanus or botulism; dermatomyositis (inflammation of several skeletal muscles in association with characteristic changes in the skin)

- ; trauma to the vagus nerves (nerve that goes to the throat, voice box, windpipe, esophagus, gastrointestinal tract, and other organs); possible association between paralysis of the voice box or larynx (known as “laryngeal paralysis”) and megaesophagus has been identified
- Esophageal blockage or obstruction: congenital (present at birth) abnormal development of major arteries of the heart, resulting in the esophagus being trapped by the blood vessels causing obstruction (abnormal arteries known as a “vascular ring anomaly”); cancer (primary esophageal cancer or cancer involving tissues around the esophagus); narrowing or stricture of the esophagus; foreign body; granuloma
- Toxicity: lead, thallium, drugs that inhibit acetylcholinesterase, an enzyme in the central nervous system (drugs are known as “anticholinesterase drugs”); acrylamide
- Endocrine disease: inadequate levels of steroids produced by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”); inadequate levels of thyroid hormone (known as “hypothyroidism”)
- Miscellaneous: thymus tumor (known as “thymoma”); stomach dilates with gas and/or fluid (known as “gastric dilatation”), and subsequently rotates around its short axis (known as “volvulus”)—condition known as “gastric dilatation-volvulus” or “bloat”; hiatal hernia; inflammation of the esophagus (known as “esophagitis”)



TREATMENT

HEALTH CARE

- Therapy for underlying cause of megaesophagus should be instituted
- Most important aspects are meeting nutritional requirements and treating or preventing aspiration pneumonia
- Aspiration pneumonia may require oxygen therapy, administration of medication in a fine spray (known as “nebulization”) and efforts to dislodge secretions in the lungs and to induce coughing (known as “coupage”), fluid therapy with balanced electrolyte solution
- Animals may be recumbent and require soft bedding and should be maintained on their chests (known as “sternal recumbency”) or turned to alternate down side every 4 hours

ACTIVITY

- Depending on cause of megaesophagus, restricted activity may not be necessary

DIET

- Nutritional requirements should be calculated precisely
- Experimentation with different food consistencies is essential—liquid gruel, small meatballs, slurries made using a blender, and others may be used
- Some cases benefit from tube feedings
- Food and water bowls should be elevated (45° to 90° from floor)
- The animal should be maintained in an upright position 10 to 15 minutes after eating or drinking

SURGERY

- Surgical intervention is indicated for congenital (present at birth) abnormal development of major arteries of the heart, resulting in the esophagus being trapped by the blood vessels causing obstruction (vascular ring anomaly); opening between a bronchus (large airway) and the esophagus (known as “bronchoesophageal fistula”); some foreign bodies and other obstructive lesions; or surgical removal of the thymus (known as “thymectomy”)
- Balloon dilation (a mechanical means of stretching the tissue) is indicated for cases of narrowing of the esophagus (known as “esophageal stricture”)
- Surgical “correction” of megaesophagus is not recommended



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics for aspiration pneumonia (broad-spectrum antibiotics or antibiotic selection based on culture and sensitivity from transtracheal wash [TTW] or bronchoalveolar lavage [BAL])—TTW and BAL are techniques used to obtain samples from the airways for microscopic evaluation and bacterial culture and sensitivity)
- Specific therapy for underlying cause, if indicated: drugs to decrease the immune response (known as “immunosuppressive drugs”)—should be used with caution if pneumonia present—for immune-mediated disease; pyridostigmine for myasthenia gravis (a disorder of

neuromuscular transmission characterized by muscular weakness and excessive fatigue)

- Drugs that improve the propulsion of contents through the stomach and intestines (known as “gastrointestinal prokinetic agents,” such as cisapride, metoclopramide) —may increase tone of the muscle between the stomach and esophagus (gastroesophageal sphincter)
- Metoclopramide increases lower esophageal sphincter tone, increases stomach motility and may increase esophageal motility
- Cisapride is more effective for backward or reverse flow of stomach contents into the esophagus (known as “esophageal reflux”) than metoclopramide; however, it may slow movement of food and liquids through the esophagus (known as “slowed esophageal transit time”); may be more helpful in cats due to increased smooth muscle in the lower esophagus
- Other motility agents (such as nizatidine) have not been evaluated for esophageal motility
- H₂-blockers for inflammation of the esophagus (known as “esophagitis”): ranitidine, cimetidine, famotidine



FOLLOW-UP CARE

PATIENT MONITORING

- Chest X-rays should be repeated when aspiration pneumonia is suspected (signs suggestive of aspiration pneumonia include fever, cough, sluggishness [lethargy])
- Cases of pneumonia may require complete blood count, blood-gas analysis to evaluate oxygen and carbon dioxide levels in the blood, and bronchoalveolar lavage (BAL)—technique used to obtain samples for microscopic evaluation and bacterial culture and sensitivity from the airways
- Animals should be assessed and weighed regularly to evaluate disease progression and ensure adequate nutritional intake

PREVENTIONS AND AVOIDANCE

- If an esophageal foreign body is identified, it should be removed as quickly as possible

POSSIBLE COMPLICATIONS

- Aspiration pneumonia
- Other complications depend on underlying cause of megaesophagus

EXPECTED COURSE AND PROGNOSIS

- Congenital (present at birth) megaesophagus cases have a guarded prognosis (20% to 46% recovery)
- Miniature schnauzers may have better prognosis
- Prognosis may be improved with identification and treatment of specific cause of megaesophagus (for example, inadequate levels of steroids produced by the adrenal glands [hypoadrenocorticism], congenital (present at birth) abnormal development of major arteries of the heart, resulting in the esophagus being trapped by the blood vessels causing obstruction [vascular ring anomaly])
- Roughly 50% of cases with myasthenia gravis (a disorder of neuromuscular transmission characterized by muscular weakness and excessive fatigue) respond to therapy
- Prognosis for megaesophagus of unknown cause (idiopathic megaesophagus) as an adult-onset disease is poor
- Owner dedication is crucial to prognosis



KEY POINTS

- Most cases of megaesophagus will require lifelong therapy, even if an underlying cause is found; client dedication is important for long-term management
- Most animals with megaesophagus will succumb to aspiration pneumonia or progression of underlying disease

MELANOCYTIC TUMORS OF THE SKIN AND DIGITS



BASICS

OVERVIEW

- Melanocytes are pigment-producing cells, located in the skin; melanoblasts are the immature cells that develop or mature into melanocytes
- Melanin is the dark pigment produced by melanocytes; it is found in the skin and hair
- Melanocytic tumors are benign or cancerous (malignant) tumors, arising from melanocytes and melanoblasts (melanin-producing cells)
- “Melanoma” is another term used for tumors arising from melanocytes; melanoma frequently is used to refer to the cancerous (malignant) tumor

GENETICS

- Unknown

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dogs—Scottish terriers, Boston terriers, Airedale terriers, cocker spaniels, boxers, English springer spaniels, Irish setters, Irish terriers, chow chows, Chihuahuas, schnauzers, and Doberman pinschers
- Cats—none

Mean Age and Range

- Dogs—9 years of age
- Cats—8 to 14 years of age

Predominant Sex

- Dogs—males may be more likely to develop melanocytic tumors than females
- Cats—none

SIGNS/OBSERVED CHANGES in the ANIMAL

- Slow or rapidly growing mass
- Lameness, if digit is involved
- Pigmented or nonpigmented (known as “amelanotic”) mass, usually a single or solitary mass
- Develops anywhere, but may be more common on face, trunk, feet, and scrotum in dogs and head, digit, ear (pinna), and nose in cats
- Regional lymph nodes (that is, the lymph nodes in the area of the mass)—may be enlarged
- Advanced disease—may have trouble breathing or harsh lung sounds because of spread of the cancer into the lungs (known as “pulmonary metastasis”)

CAUSES

- Unknown

RISK FACTORS

- Unknown



TREATMENT

HEALTH CARE

- Inpatient, if undergoing surgery
- Fluid administration—indicated during surgery

- Melanoma of the digit—may require bandaging of the limb after surgery

ACTIVITY

- Depends on location of tumor
- Generally, restrict until sutures are removed

DIET

- Normal

SURGERY

- Wide surgical excision—treatment of choice
- Amputation of digit—if nail bed or digit affected



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Chemotherapy—recommended if surgical removal is incomplete; the tumor is not able to be removed surgically (known as a “nonresectable tumor”); or spread of the cancer (metastasis) has occurred
- Dacarbazine (DTIC) (dogs), doxorubicin, and carboplatin—reported to induce partial and complete remission in a small number of animals; may be drugs of choice
- Cimetidine—shown to be of some benefit in horses and people with malignant melanoma; believed to act as a biologic response modifier by reversing suppressor T-cell-mediated immune suppression; has not been evaluated for this purpose in dogs and cats
- Vaccination with xenogeneic human tyrosinase (a DNA vaccine) in dogs with advanced melanoma shows promise in experimental clinical trials



FOLLOW-UP CARE

PATIENT MONITORING

- Evaluate for evidence of recurrence and spread (metastasis)—1, 3, 6, 9, 12, 18, 21, and 24 months after surgery; if the owner believes the mass is returning; if the patient is otherwise not normal
- Chest X-rays—at the time of rechecks and periodically thereafter

EXPECTED COURSE AND PROGNOSIS

- Malignant melanoma may spread (metastasize) early in the course of the disease; thus prognosis is guarded
- Dogs—25% to 50% of melanomas reported to be cancer (malignant); melanomas on the digit, footpad, and scrotum have a greater likelihood of being malignant
- Mean survival with benign melanomas (dogs) of the skin is greater than 24 months
- Survival with malignant melanoma (dogs)—skin: 8 to 18 months; digit: 12 to 16.9 months
- Cats—35% to 50% of melanomas reported to be malignant
- Mean survival with melanoma of the skin or digit (cats)—not frequently reported; 4.5 months after surgery in one study of 57 cats



KEY POINTS

- Need for early surgical removal
- Do not take a “wait-and-see” approach
- Malignant melanoma may spread (metastasize) early in the course of the disease; thus prognosis is guarded

GRANULOMATOUS MENINGOENCEPHALOMYELITIS

(INFLAMMATION OF THE BRAIN, SPINAL CORD, AND MENINGES)



BASICS

OVERVIEW

- “Granulomatous” refers to inflammation characterized by the presence of nodules; “meningoencephalomyelitis” is inflammation of the brain, spinal cord and their surrounding membranes (the membranes are known as “meninges”)
- Granulomatous meningoencephalomyelitis (also known as “GME”) refers to an inflammatory disease that affects the central nervous system; it can be localized, widespread, or involve multiple locations
- Confirmation of the disease diagnosis is only possible through microscopic analysis of the affected nervous tissue, obtained through biopsy
- Granulomatous meningoencephalomyelitis (GME) is the most accepted and recognized central nervous system inflammatory disorder in the dog
- Many less serious viral and idiopathic disorders frequently are diagnosed erroneously as being “granulomatous meningoencephalomyelitis (GME)” (“idiopathic” means the disease is of unknown cause)
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae

GENETICS

- A genetic factor is not proven

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Historically, granulomatous meningoencephalomyelitis (GME) has been considered a disease of young to middle-age, toy-breed dogs (especially terriers and poodles); however, any medium and larger breed can be affected

Mean Age and Range

- Mean—5 years of age
- Range, 6 months to 10 years

Predominant Sex

- Both sexes can be affected, but females have slightly a higher number of cases than do males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs depend on the form of the disease and location of nerve tissue involved
- The cerebral form of disease (involving the brain) frequently results in seizure activity

Ocular form

- Sudden (acute) onset of blindness, with dilated and unresponsive pupils; the “pupil” is the circular or elliptical opening in the center of the iris of the eye; the “iris” is the colored or pigmented part of the eye

Focal form

- Cerebral lesions (involving the brain)—disorientation, behavioral changes, seizures, blindness, compulsive circling, head pressing
- Brainstem lesion (involving the part of the brain that is connected to the spinal cord that controls functions like breathing and heart rate)—drowsiness or sleepiness (known as “somnia”), cranial nerve deficits (most commonly facial nerve deficit and lack of control of equilibrium, balance, and orientation [known as “vestibular dysfunction”]), one-sided weakness or partial paralysis (known as “hemiparesis”)
- Spinal cord lesion—weakness or partial paralysis of all four legs (known as “tetraparesis”) for lesions in the spinal cord involving the area of the first cervical vertebra through the fifth cervical vertebra (C₁ through C₅) or the sixth cervical vertebra through the second thoracic vertebra (C₆ through T₂) or weakness or partial paralysis of the rear legs (known as “paraparesis”) for lesions in the spinal cord involving the area of the third thoracic vertebra through the third lumbar vertebra (T₃ through L₃) or the fourth lumbar vertebra through the second sacral vertebra (L₄ through S₂) and a wobbly, incoordinated or “drunken” appearing gait or movement (known as “

ataxia”)

CAUSES

- Unknown

RISK FACTORS

- Unknown
- Some dogs develop clinical signs within 5 to 10 days of receiving vaccinations



TREATMENT

HEALTH CARE

- Stable patients can be discharged with recommended treatment
- Inpatient—for severely affected dogs; monitor patient closely for progression of nervous system deficits
- Intravenous fluids for patients that have lack of appetite (known as “anorexia”); fluids should be administered carefully to avoid overhydration and worsening of fluid build-up in the brain (known as “cerebral edema”)
- Provide a padded cage for dogs with lack of control of equilibrium, balance, and orientation (vestibular dysfunction), severe dementia or seizure activity
- Recumbent patients should be turned frequently (every 4 hours) to avoid pressure sores and lung congestion

ACTIVITY

- Depends on the severity of disease and location of nerve tissue involved
- Patients with a wobbly, incoordinated or “drunken” appearing gait or movement (ataxia) should be confined to a padded cage to avoid injury

DIET

- Ensure adequate caloric intake



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids—dexamethasone followed by prednisone; the steroid dose should be adjusted by your pet’s veterinarian, according to response to treatment and side effects—the goal is to find the dose that keeps the clinical signs controlled with minimal side effects
- To prevent gastrointestinal ulceration, administer famotidine (an H₂-blocker to reduce stomach acid) with the steroid therapy
- Phenobarbital to control seizures
- Chemotherapeutic drugs used to decrease the immune response—azathioprine, cytosine arabinoside, or cyclophosphamide
- Medications to decrease the immune response (known as “immunosuppressive drugs”)—cyclosporine, leflunomide
- Radiation therapy is an alternative treatment in the focal form of the disease when other therapies have failed (the diagnosis should be confirmed by microscopic evaluation of tissue samples obtained by biopsy, before starting radiation therapy)



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat nervous system examination periodically (every 2 to 4 weeks)
- Evaluate blood work (complete blood count [CBC] and biochemical profile) regularly to monitor for low white-blood cell count (known as “leukopenia”), low platelet count (known as “thrombocytopenia”), and liver and kidney function
- Monitor urine in patients on long-term steroid treatment—protein in the urine (known as “proteinuria”) or urinary tract infection are

frequent consequences of long-term steroid treatment

POSSIBLE COMPLICATIONS

- Deterioration of clinical signs, despite aggressive treatment
- Repeated or prolonged seizure activity (known as “status epilepticus”)
- Dementia
- Brain pushes downward in the skull and herniates through the opening that leads to the neck (known as “tentorial herniation” or “brain herniation”) and death

EXPECTED COURSE AND PROGNOSIS

- Not all patients with central nervous system inflammatory disease have a poor prognosis
- Granulomatous meningoencephalomyelitis (GME) has been characterized as a fatal disease without enough scientific evidence; it is uncertain if dogs that survive inflammatory central nervous system disease had GME, as brain biopsies rarely are obtained to provide a definitive diagnosis



KEY POINTS

- Clinical signs overlap significantly among different central nervous system inflammatory diseases—a diagnostic work-up is very important
- Mortality rate for granulomatous meningoencephalomyelitis (GME) is clearly biased by the severe cases that go to postmortem examinations; brain biopsies rarely are obtained during life to provide a definitive diagnosis and to provide a more accurate mortality rate
- Some dogs with central nervous system inflammatory disease can be treated successfully; however, long-term treatment and client compliance are required
- Steroid therapy may be necessary for a prolonged period or for life

METALDEHYDE POISONING



BASICS

OVERVIEW

- Metaldehyde—an ingredient of slug and snail baits; used as solid fuel for some camp stoves
- Baits—liquids, granules, wettable powders, or pelleted baits (pellets often mixed with grain); baits also may contain other poisons (such as arsenate or insecticides)
- Metaldehyde poisoning primarily affects the nervous system

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs (most common) and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- May occur immediately after ingestion of metaldehyde or may be delayed for up to 3 hours
- Anxiety and panting are early signs
- Excessive salivation/drooling (known as “hypersalivation”) and/or vomiting or diarrhea may occur
- Wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)
- Muscle tremors
- Seizures—may be intermittent early, but progress to continuous seizure activity; not necessarily set off by external stimuli
- Between seizures—may note muscle tremors and anxiety; may be overly sensitive to sounds, light, and/or touch
- Markedly elevated body temperature (known as “hyperthermia”)—temperature up to 42.2° C (108° F) common; probably caused by excessive muscle activity from seizures; hyperthermia may lead to a blood-clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”) or multiple organ failure, if uncontrolled
- Rapid heart rate (known as “tachycardia”) and deeper and more rapid breathing (known as “hyperpnea”) than normal
- Short, rapid movements of the eyeball (known as “nystagmus”) or dilated pupils (known as “mydriasis”) are possible; the “pupil” is the circular or elliptical opening in the center of the iris of the eye; the “iris” is the colored or pigmented part of the eye

CAUSES

- Ingestion of metaldehyde

RISK FACTORS

- Living in area with a high number of snails and slugs
- Metaldehyde poisoning is found more commonly in coastal and low-lying areas, which have a higher number of snails and slugs than other areas



TREATMENT

HEALTH CARE

- Emergency inpatient intensive care, until seizures cease and elevated body temperature (hyperthermia) is controlled
- Monitor to prevent aspiration of vomitus
- Fluids often are necessary to treat dehydration or possibly acidosis (a condition in which levels of acid are increased in the blood)

ACTIVITY

- Restricted, so that patient does not injure itself during seizures

DIET

- Do not feed patients that are vomiting, having seizures, or are sedated heavily



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No antidote is available for metaldehyde poisoning
- The veterinarian will attempt to decrease absorption of metaldehyde in patients that have no clinical signs or that have been stabilized with medications to induce vomiting (known as “emetics”), flushing the stomach (known as “gastric lavage”), and/or administration of activated charcoal, as appropriate
- Seizures may be controlled with diazepam, barbiturates, and/or gas anesthesia; in addition, a muscle relaxant (methocarbamol) may be administered



FOLLOW-UP CARE

PATIENT MONITORING

- Periodically allow sedatives or anesthetics to wear off to re-evaluate seizure activity

PREVENTIONS AND AVOIDANCE

- Do not apply metaldehyde in areas accessible to pets
- Some manufacturers dye the product green or blue to assist with identification
- Some states require manufacturers to adjust the formulation to decrease the tastiness (palatability) to pets

POSSIBLE COMPLICATIONS

- Liver or kidney dysfunction are possible several days after recovery from the initial signs and probably are sequelae to the seizures and elevated body temperature (hyperthermia)
- Aspiration pneumonia is a concern with any patient that has seizures
- Elevated body temperature (hyperthermia) may lead to a blood-clotting disorder (disseminated intravascular coagulopathy or DIC) or multiple organ failure
- Temporary blindness or memory loss may occur

EXPECTED COURSE AND PROGNOSIS

- Prognosis—principally depends on the amount of metaldehyde ingested, time to treatment, and quality of care
- Delayed or non-aggressive treatment may result in death within hours of exposure



KEY POINTS

- Do not apply metaldehyde in areas accessible to pets
- Metaldehyde poisoning is found more commonly in coastal and low-lying areas, which have a higher number of snails and slugs than other areas
- Emergency inpatient intensive care is necessary, until seizures cease and elevated body temperature (hyperthermia) is controlled
- No antidote is available for metaldehyde poisoning
- Delayed or non-aggressive treatment may result in death within hours of exposure

MYASTHENIA GRAVIS



BASICS

OVERVIEW

- A disorder of signal transmission between nerves and muscles (known as “neuromuscular transmission”), characterized by muscular weakness and excessive fatigue

GENETICS

- Congenital (present at birth) familial (runs in families or lines) forms—Jack Russell terriers, English springer spaniels, smooth fox terriers; smooth-haired miniature dachshunds, autosomal recessive mode of inheritance
- Acquired (not inherited, present later in life/after birth)—as with other autoimmune diseases, requires appropriate genetic background for disease to occur; multiple factors involved, including environmental, infectious, and hormonal influences
- Familial (runs in families or lines) forms of acquired myasthenia gravis occur in the Newfoundland and Great Dane

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Congenital (present at birth)—Jack Russell terriers; English springer spaniels; smooth fox terriers, smooth-haired miniature dachshunds
- Acquired (not inherited, present later in life/after birth)—several dog breeds: golden retrievers; German shepherd dogs; Labrador retrievers; dachshunds; Scottish terriers; Akitas and cat breeds: Abyssinian and Somali

Mean Age and Range

- Congenital (present at birth)—6 to 8 weeks of age
- Acquired (not inherited, present later in life/after birth)—bimodal age of onset; dogs: 1 to 4 years of age and 9 to 13 years of age

Predominant Sex

- Congenital (present at birth)—none
- Acquired (not inherited, present later in life/after birth)—may be a slight susceptibility for females in the young age group; none in the old age group (as described in **Mean Age and Range**)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Acquired (not inherited, present later in life/after birth)—may have several clinical presentations ranging from localized involvement of the muscles of the esophagus (the tube running from the throat to the stomach), muscles of the throat (pharynx), and muscles adjacent to the eye (known as “extraocular muscles”) to acute generalized collapse
- Any dog with acquired megaesophagus (enlargement of the esophagus), loss of normal reflexes (known as “lower motor neuron weakness”), or a mass in the front central area of the chest (known as a “cranial mediastinal mass”) should be evaluated for myasthenia gravis
- Regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth)—common; important to differentiate between vomiting (forceful ejection of stomach contents up through the esophagus and mouth) and regurgitation
- Voice change
- Exercise-related weakness
- Acute collapse
- Progressive weakness
- Sleep with eyes open
- May look normal when at rest
- Excessive drooling, repeated attempts at swallowing
- Loss of muscle mass (known as “muscle atrophy”)—usually not found
- Difficulty breathing (known as “dyspnea”)—with aspiration pneumonia
- Fatigue or cramping—with mild exercise
- Subtle nervous system findings: decreased or absent blink reflex (known as “palpebral reflex”); may note a poor or absent gag reflex; spinal reflexes usually normal but may fatigue (rarely absent and dog unable to support its weight)
- Abnormal position of the neck (known as “ventroflexion”)—cats, uncommon in dogs

CAUSES

- Congenital (present at birth)
- Immune-mediated disease
- Secondary to cancer (known as “paraneoplastic”)

RISK FACTORS

- Appropriate genetic background
- Tumor or cancer—particularly thymus tumor (known as “thymoma”)
- Methimazole treatment (cats)—may result in reversible disease
- Vaccination can exacerbate active myasthenia gravis
- Intact female



TREATMENT

HEALTH CARE

- Inpatient—until adequate dosages of drugs that inhibit acetylcholinesterase, an enzyme in the central nervous system (drugs are known as “anticholinesterase drugs”), are achieved
- Aspiration pneumonia—may require intensive care
- Feeding tube—may be required if patient is unable to eat or drink without significant regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth)
- Oxygen therapy, intensive antibiotic therapy, intravenous fluid therapy, and supportive care—generally required for aspiration pneumonia
- Nutritional maintenance with a feeding tube—multiple feedings of a high-caloric diet; good hygiene care
- Elevation of food and water bowls

ACTIVITY

- Self-limited, owing to the severity of muscle weakness and extent of aspiration pneumonia

DIET

- May try different consistencies of food—gruel; hard food; soft food; evaluate what is best tolerated

SURGERY

- Cranial mediastinal mass—thymus tumor (thymoma)
- Before attempting surgical removal, stabilize patient with drugs (anticholinesterase drugs) that inhibit acetylcholinesterase, an enzyme in the central nervous system, and treat aspiration pneumonia
- Weakness may not be clinically evident initially
- Suspected thymus tumor (thymoma)—test all patients for acquired myasthenia gravis before surgery



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drugs that inhibit acetylcholinesterase, an enzyme in the central nervous system (anticholinesterase drugs)—prolong the action of acetylcholine (a chemical that transmits messages between nerves and muscles) at the neuromuscular junction; pyridostigmine bromide syrup (Mestinon® syrup)
- Steroids—initiated if poor response to pyridostigmine or if no response to edrophonium chloride challenge
- Azathioprine, a chemotherapeutic drug used to decrease the immune response



FOLLOW-UP CARE

PATIENT MONITORING

- Return of muscle strength should be evident
- Chest X-rays—evaluated every 4 to 6 weeks for resolution of enlarged esophagus (megaesophagus)
- Acetylcholine receptor (AChR) antibody titers—evaluated every 6 to 8 weeks; decrease to the normal range with clinical remission

POSSIBLE COMPLICATIONS

- Aspiration pneumonia
- Breathing may stop (known as “respiratory arrest”)

EXPECTED COURSE AND PROGNOSIS

- No severe aspiration pneumonia or weakness of the throat (pharynx)—good prognosis for complete recovery; resolution usually within 6 to 8 months.
- Thymus tumor (thymoma) present—guarded prognosis, unless complete surgical removal and control of myasthenic signs are achieved



KEY POINTS

- Although the disease is treatable, most patients require months of special feeding and medication
- A dedicated owner is important to a favorable outcome for acquired myasthenia gravis

MYCOPLASMOSIS: INFECTIOUS DISEASES CAUSED BY *MYCOPLASMA*, *UREAPLASMA*, OR *ACHOLEPLASMA*



BASICS

OVERVIEW

- “Mycoplasmosis” is the general name for diseases caused by three groups of infectious agents: *Mycoplasma*, *T-mycoplasma* or *Ureaplasma*, and *Acholeplasma*; each of these infectious agents are gram-negative bacteria that can live and grow in the absence of oxygen (known as “anaerobic” bacteria); they lack cell walls
- They are found everywhere in nature; many cause disease in people, animals, plants, and insects

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- All ages

SIGNS/OBSERVED CHANGES in the ANIMAL

- Simultaneous inflammation of several joints (known as “polyarthritis”)—long-term (chronic) intermittent lameness; reluctance to move; joint pain; joint swelling; and generalized build-up of fluid under the skin of the legs (known as “diffuse limb edema”)
- Fever
- General signs of discomfort and “not feeling well” (known as “malaise”)
- Inflammation of the moist tissues of the eye (known as “conjunctivitis”)—may involve one or both eyes
- Squinting or spasmodic blinking (known as “blepharospasm”); fluid build-up (known as “edema”) of the moist tissue covering of the eyeball, around the cornea (condition known as “chemosis”); reddening of the moist tissues of the eye; overflow of tears (known as “epiphora”); discharge from the eyes, which may be clear or may contain pus
- Mild inflammation of the nose (known as “rhinitis”)—sneezing
- Other signs are related to the site of infection—in dogs, may see signs of pneumonia and upper respiratory infections; urinary and genital tract infections (such as inflammation of the prostate, bladder, or the inner lining of the uterus); inflammation of the colon (known as “colitis”) and in cats, may see signs of pneumonia; urinary tract infections; abortions, and long-term (chronic) skin abscesses

CAUSES

- *Mycoplasma* of dogs—*M. canis*, *M. spumans*, *M. maculosum*, *M. edwardii*, *M. cynos*, *M. molare*, *M. opalescens*, *M. feliminutum*, *M. gateae*, *M. arginini*, *M. bovigentialium*, *Acholeplasma laidlawii*, and ureaplasmas
- *Mycoplasma* of cats—*M. felis*, *M. gateae*, *M. feliminutum*, *M. arginini*, *M. pulmonis*, *M. arthritidis*, *M. gallisepticum*, *Acholeplasma laidlawii*, and ureaplasmas

RISK FACTORS

- Generalized (systemic) infection associated with an inability to develop a normal immune response (known as “immunodeficiency”); suppression of immune response, as by drugs (known as “immunosuppression”); or cancer
- Impaired resistance of the host—may allow the organism to cross the protective, mucosal barrier and spread into the body
- Predisposing factors—stresses (such as reproductive problems associated with overcrowded operations) and other factors (such as tumors or stones in the urinary tract)



TREATMENT

HEALTH CARE

- Outpatient



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Sensitive to certain antibiotics, such as tetracycline, doxycycline, chloramphenicol
- No standardized procedure is available for bacterial culture and susceptibility tests for these infectious agents
- Topical (directly applied to the eye) antibiotic—for inflammation of the moist tissues of the eye (conjunctivitis)
- Other antibiotics that may be used include gentamicin, kanamycin, spectinomycin, spiramycin, tylosin, erythromycin, nitrofurans, and fluoroquinolones



FOLLOW-UP CARE

PATIENT MONITORING

- Treat for an extended period of time

PREVENTIONS AND AVOIDANCE

- No vaccines are available to prevent infection
- Organism readily killed by drying, sunshine, and chemical disinfection

EXPECTED COURSE AND PROGNOSIS

- Prognosis good in animals with normal immune systems and given appropriate antibiotic therapy



KEY POINTS

- “Mycoplasmosis” is the general name for diseases caused by three groups of infectious agents: *Mycoplasma*, *T-mycoplasma* or *Ureaplasma*, and *Acholeplasma*; each of these infectious agents are gram-negative bacteria that can live and grow in the absence of oxygen (known as “anaerobic” bacteria)
- They are found everywhere in nature; many cause disease in people, animals, plants, and insects
- Prognosis good in animals with normal immune systems and given appropriate antibiotic therapy

MULTIPLE MYELOMA



BASICS

OVERVIEW

- Plasma cells are specialized white-blood cells; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease
- A “clonal population of cells” is a group of cells descended from a single cell; all of the cells have the same genetic make-up
- Multiple myeloma is an uncommon cancer derived from a clonal population of cancerous (malignant) plasma cells in the bone marrow
- Three of four defining features must be present for diagnosis of multiple myeloma: immune protein from a single clone of cells (known as a “monoclonal gammopathy”), seen as a spike in the gamma region of a protein analysis (known as a “protein electrophoresis”) of blood; cancerous (malignant) plasma cells or high number of plasma cells in the bone marrow (known as “plasmacytosis”); destruction of areas of bone (known as “lytic bone lesions”); and a particular type of protein found in the urine (known as “Bence Jones [light-chain] proteinuria”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- German shepherd dogs and other purebred dogs more often than mixed-breed dogs

Mean Age and Range

- Primarily middle-aged or old dogs and cats (6 to 13 years)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Attributed to bone infiltration and destruction of bone (lysis), effects of proteins produced by the tumor (such as increased protein in the blood leading to sludging of the blood [known as “hyperviscosity”] and kidney damage), and infiltration of organ(s) by cancerous cells
- Depend on location and extent of disease
- Weakness
- Lameness
- Pain
- Partial paralysis (known as “paresis”)
- Urinary incontinence
- Bleeding from the nose (known as “epistaxis”)—may involve one or both nostrils
- Bleeding in the back part of the eye (known as “retinal hemorrhage”) and blindness
- Bleeding from needle punctures to collect blood or to administer intravenous medications and/or fluids
- Dementia
- General discomfort or uneasiness (known as “malaise”)
- Labored breathing
- Increased urination (known as “polyuria”)
- Increased thirst (known as “polydipsia”)
- Bleeding involving the gastrointestinal tract

Dogs

- Bleeding—especially from the nose or mucous membranes (the moist tissues of the mouth, eyes, and other areas of the body)—seen in 36% of affected dogs
- Blindness, retinal hemorrhage, or dilated retinal vessels (35%); detached retina; glaucoma; inflammation of the front part of the eye, including the iris (known as “anterior uveitis”)
- Lameness (47%), bone pain and weakness (60%)—with destruction of areas of bone (lytic bone lesions)
- Dementia, generalized discomfort or uneasiness (malaise)—seen in 11% of affected dogs; and coma (rare)
- Increased thirst (polydipsia) and increased urination (polyuria)—seen in 25% of affected dogs—with increased levels of calcium in the blood (known as “hypercalcemia”) or kidney dysfunction
- Pale gums and other moist tissues of the body (mucous membranes)

- Fever
- Sluggishness (lethargy)
- Enlarged liver and spleen (known as “hepatosplenomegaly”)

Cats

- Lack of appetite (known as “anorexia”)
- Weight loss
- Generalized discomfort or uneasiness (malaise)
- Increased thirst (polydipsia)
- Increased urination (polyuria)
- Fever

CAUSES

- Unknown



TREATMENT

HEALTH CARE

- Consult a veterinary oncologist for latest information regarding treatment
- Inpatient treatment if animal has excessive levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”), high levels of calcium in the blood (hypercalcemia), a bleeding disorder or clinically important bacterial infection
- Plasmapheresis (medical process in which whole blood is removed from the body, the blood cells are separated from the fluid portion of the blood and then are put into a sterile fluid and transfused back into the body), when available, lowers protein burden
- Patient with signs of increased protein in the blood leading to sludging of the blood (hyperviscosity)—the veterinarian may perform phlebotomy (a medical procedure in which an incision is made into the vein, for the purpose of withdrawing blood) and replace the volume of blood withdrawn intravenously with an equal volume of fluids
- Radiation therapy may be used on isolated areas with the goal of cure (known as “curative intent”) or to control signs and improve the patient’s condition, but not to cure (known as “palliative intent”)
- Use sterile technique and be prepared to control bleeding from any site used for obtaining a blood sample or for administering intravenous medication of fluids
- Bacterial infection—treat aggressively with appropriate antibiotics
- High levels of calcium in the blood (hypercalcemia) and kidney failure—treat appropriately
- Affected animals may have low numbers of neutrophils or nonfunctional lymphocytes (neutrophils and lymphocytes are types of white-blood cells); take care to minimize exposure to infectious agents (such as viruses, bacteria, and fungi)
- Use sterile or very clean technique when performing any invasive techniques

ACTIVITY

- Treat animal as being unable to develop a normal immune response (known as “immune compromised”); take care to prevent bacterial infection (such as caused by puncture wounds from dog or cat fights)

DIET

- Dietary changes may be necessary, if animal is in kidney failure

SURGERY

- Areas nonresponsive to chemotherapy or single (solitary) lesions may be removed surgically



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Chemotherapy is intended to improve the patient’s condition, but not to cure the multiple myeloma (palliative treatment), but long remissions are possible

- Dogs—melphalan and prednisone; cyclophosphamide can be used in addition to or in place of melphalan
- Liposome-encapsulated doxorubicin was reported to be effective in one dog
- Cats—melphalan and prednisone
- Cyclophosphamide—may be beneficial to substitute for melphalan with animals that have low platelet or thrombocyte counts (known as “thrombocytopenia”)
- Dogs—more aggressive combination chemotherapy protocol; cyclophosphamide, vincristine, melphalan, and prednisone



FOLLOW-UP CARE

PATIENT MONITORING

- Complete blood count (CBC) and platelet count—weekly for at least 4 weeks to assess bone-marrow response to chemotherapeutic drugs
- Blood tests with abnormal results should be repeated monthly to evaluate response to treatment
- Protein analysis of blood (protein electrophoresis) monthly for several months, until normal protein patterns are obtained, then monitor periodically for relapse
- Abnormal skeletal X-rays should be repeated monthly, then every other month until normal to evaluate response to treatment

POSSIBLE COMPLICATIONS

- Bleeding
- Secondary infections
- Fractures occurring at the site of weakened bone (known as “pathologic fractures”), due to the presence of multiple myeloma
- Chemotherapy may cause low white-blood cell counts (known as “leukopenia”) or low platelet or thrombocyte counts (thrombocytopenia); lack of appetite (anorexia); hair loss (known as “alopecia”); bloody inflammation of the bladder (known as “hemorrhagic cystitis”); and/or inflammation of the pancreas (known as “pancreatitis”)

EXPECTED COURSE AND PROGNOSIS

- Even with treatment, it may be several months before clinical signs resolve
- Continuous care must be taken to protect patients from secondary infection

Dogs

- Median survival with chemotherapeutic agents and prednisone—18 months
- Median survival with prednisone—7 months
- Complete response in 43%; partial response in 49%
- High levels of calcium in the blood (hypercalcemia), extensive destruction of areas of bone (bone lysis), or presence of a particular type of protein in the urine (Bence Jones proteinuria) often shorter survival

Cats

- Survival with chemotherapeutic agents and prednisone—2 to 9 months



KEY POINTS

- Chemotherapy is intended to improve the patient’s condition, but not to cure the multiple myeloma (palliative treatment), but long remissions are possible
- Relapse will occur
- Side effects are determined by the drugs used
- Most patients develop mild low white-blood cell counts (leukopenia) with chemotherapy

MYELOPATHY—PARESIS AND PARALYSIS IN CATS

(DISORDER OF THE SPINAL CORD LEADING TO WEAKNESS AND PARALYSIS IN CATS)



BASICS

OVERVIEW

- “Myelopathy”—any disorder or disease affecting the spinal cord; depending upon the location of the lesion, a myelopathy can cause weakness or partial paralysis (known as “paresis”) or complete loss of voluntary movements (known as “paralysis”)
- Paresis or paralysis may affect all four limbs (known as “tetraparesis” or “tetraplegia,” respectively), may affect only the rear legs (known as “paraparesis” or “paraplegia,” respectively), the front and rear leg on the same side (known as “hemiparesis” or “hemiplegia,” respectively) or only one limb (known as “monoparesis” or “monoplegia,” respectively)
- Paresis and paralysis also can be caused by disorders of the nerves and/or muscles to the legs (known as “peripheral neuromuscular disorders”)
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae
- The brainstem is the part of the brain that is connected to the spinal cord that controls functions like breathing and heart rate

GENETICS

- Lysosomal storage diseases (inherited metabolic diseases in which harmful levels of materials accumulate in the body’s cells and tissues)—examples include gangliosidosis GM1/GM2, sphingomyelinosis (Niemann-Pick disease), mucopolysaccharidosis VI, and glycogenosis type IV that cause weakness (paresis) or paralysis in cats—they have an autosomal recessive pattern of inheritance
- Disorder characterized by progressive changes in the part of the nerve that carries impulses away from the nerve cell body and toward the muscles (known as “neuroaxonal dystrophy”) and disorder characterized by loss of nerve cell bodies in the brainstem and spinal cord (known as “spinal muscular atrophy”)—degenerative diseases of the spinal cord reported in cats as autosomal recessive conditions
- Disorders characterized by a cavity in the spinal cord (known as “syringohydromyelia”) or defective development of the spinal cord (known as “myelodysplasia”) may be associated with congenital (present at birth) defects of the spine and spinal cord (known as “sacrocaudal dysgenesis”), an autosomal dominant condition in the Manx

SIGNALMENT/DESCRIPTION OF ANIMAL

Species

- Cats

Breed Predispositions

- Gangliosidosis GM1 type 2—Siamese and Korat
- Glycogen storage disease type IV—Norwegian Forest cats
- Syringohydromyelia/myelodysplasia—Manx with sacrocaudal dysgenesis
- Peripheral and central distal axonopathy—Birman

SIGNS/OBSERVED CHANGES in the ANIMAL Cervical syndrome (spinal lesion is located in the neck)—weakness of all four legs (tetraparesis) or paralysis of all four legs (tetraplegia); weakness of the front and rear leg on one side of the body (hemiparesis) or paralysis of the front and rear leg on one side of the body (hemiplegia); wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”); abnormalities in which the normal subconscious awareness of the location of the legs and movement is altered (known as “proprioceptive deficits”); normal reflexes or exaggerated reflexes (known as “hyperreflexia”); the cat may be overly sensitive to pain or touch (known as “hyperesthesia”) around the neck and Horner’s syndrome (condition in which one pupil is small or constricted, the eyelid droops, and the eyeball is withdrawn into the socket) also may be present

● Cervicothoracic syndrome (spinal lesion involves the area of the lower part of the neck and/or the chest)—weakness of all four legs (tetraparesis) or paralysis of all four legs (tetraplegia); weakness of the front and rear leg on one side of the body (hemiparesis) or of the front and rear leg on one side of the body (hemiplegia); wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”); abnormalities in which the normal subconscious awareness of the location of the legs and movement is altered (known as “proprioceptive deficits”); decreased reflexes (known as “hyporeflexia”) or lack of reflexes (known as “areflexia”), decreased muscle tone (known as “hypotonus”), and loss of muscle mass (known as “muscle atrophy”) on one or both front legs and normal reflexes or exaggerated reflexes (known as “hyperreflexia”) and increased muscle tone (known as “hypertonus”) of the rear legs; the cat may be overly sensitive to pain or touch (hyperesthesia) along the spine around the area of the junction of the neck and chest; and Horner’s

syndrome (condition in which one pupil is small or constricted, the eyelid droops, and the eyeball is withdrawn into the socket) also may be present

- Thoracolumbar syndrome (spinal lesion involves the area of the spine from the end of the ribs to the pelvis)—weakness of the rear legs (paraparesis) or paralysis of the rear legs (paraplegia); wobbly, incoordinated or “drunken” appearing gait or movement (ataxia) of the rear legs; abnormalities in which the normal subconscious awareness of the location of the rear legs and movement is altered (proprioceptive deficits); increased muscle tone (hypertonus) and normal reflexes or exaggerated reflexes (hyperreflexia) of the rear legs; the cat may be overly sensitive to pain or touch (hyperesthesia) along the spine at the area from the end of the ribs to the pelvis; the cat may have absent or decreased sensation below the spinal lesion; lack of control or urination (known as “urinary incontinence”) also may be present—urinary incontinence is characterized by a tense bladder that is difficult to express (so called “upper motor neuron bladder”)

- Lumbosacral syndrome (spinal lesion involves the area of the spine at the pelvis)—weakness of the rear legs (paraparesis) or paralysis of the rear legs (paraplegia); weakness of one rear leg (monoparesis) or paralysis of one rear leg (monoplegia); mild rear leg wobbly, incoordinated, or “drunken” appearing gait; abnormalities in which the normal subconscious awareness of the location of the rear legs and movement is altered (proprioceptive deficits); decreased reflexes (known as “hyporeflexia”) or lack of reflexes (areflexia); decreased muscle tone (hypotonus); and loss of muscle mass (muscle atrophy) of the rear leg(s); the cat may be overly sensitive to pain or touch (hyperesthesia) along the spine around the area of the pelvis; the cat may have decreased or absent sensation below the lesion as well as decreased or absent tail and anal tone; lack of ability to control urination (urinary incontinence) and bowel movements (known as “fecal incontinence”) also may be present—urinary incontinence is characterized by large and flaccid bladder that is easy to express (so called “lower motor neuron bladder”)

CAUSES

- Degenerative disease of the spinal cord
- Inherited disease of the spinal cord
- Abnormal structural development of the backbones (vertebrae) or spinal cord
- Metabolic abnormalities
- Tumors or cancer (such as lymphoma, osteosarcoma, fibrosarcoma, and cancer that has spread to the spine/spinal cord [known as “metastatic cancer”])
- Inflammatory or infectious disease (such as feline infectious peritonitis [FIP], toxoplasmosis, and feline leukemia virus (FeLV)-associated disorder of the spinal cord [known as “FeLV-associated myelopathy”])
- Trauma (such as fractures or dislocations of the backbones [vertebrae] and penetrating wounds [bite wounds, BB pellets])
- Abnormal blood flow to the spine/spinal cord

RISK FACTORS

- Outdoor cats—at risk for traumatic and infectious inflammation of the spinal cord (known as “myelitis”)
- Feline leukemia virus (FeLV)-positive cats—at risk for lymphoma



TREATMENT

HEALTH CARE

- Emergency evaluation and possible surgery—when a traumatic cause of weakness (paresis)/paralysis is suspected
- Inpatient medical management—for severe nervous system deficits, such as paralysis and lack of control of urination (urinary incontinence)
- Cats that cannot walk (known as being “nonambulatory”) should be confined to a soft padded crate or enclosed area, they should be kept dry and clean and turned every 6 hours, if they are unable to stay lying on their chests
- If the cat has lack of control of urination (urinary incontinence), the bladder should be emptied every 6 to 8 hours
- Prevent/treat “bed sores” (known as “decubital ulcers”) and skin lesions that develop due to contact with urine, when the hair and skin remain damp (known as “urine scald”)
- Treat constipation
- Physical therapy is useful to prevent loss of muscle mass (muscle atrophy) and to keep joints flexible, especially for postoperative rehabilitation

ACTIVITY

- Restricted—especially when a traumatic cause of weakness (paresis)/paralysis is suspected, but also to prevent spinal trauma secondary to the paresis/paralysis

SURGERY

- Surgical management—for fractures or dislocations of the backbones (vertebrae), intervertebral disk disease, and some tumors or

cancer



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Not recommended until a diagnosis has been established
- For spinal trauma—methylprednisolone administered within 8 hours from the time of trauma
- Antibiotics, if the cat develops a urinary tract infection



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat nervous system examination—the frequency of the re-examinations should be determined based on the severity and progression of the cat's nervous system status

PREVENTIONS AND AVOIDANCE

- Depend on underlying cause
- Keep cat indoors or on leash when outside to avoid spinal trauma

POSSIBLE COMPLICATIONS

- Urinary tract infection
- Skin lesions that develop due to contact with urine, when the hair and skin remain damp (urine scald)
- Constipation or lack of control of bowel movements (fecal incontinence)
- Loss of muscle mass (muscle atrophy)
- “Bed sores” (decubital ulcers)

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause



KEY POINTS

- If the cat is treated as an outpatient, it is important to understand all aspects of nursing care and possible complications; discuss the care of your cat with the veterinarian

DEGENERATIVE MYELOPATHY

(A SPINAL CORD DISEASE)



BASICS

OVERVIEW

- “Degenerative” refers to degeneration; “degeneration” is the decline or loss of function or structure of a tissue or organ; “myelopathy” is a disorder of the spinal cord
- “Degenerative myelopathy” is a disease of the spinal cord that causes progressive weakness of the hind limbs (known as “paraparesis”)

GENETICS

- Unknown inheritance pattern
- A familial (runs in certain families or lines of animals) inheritance currently is suspected due to the number of purebred dogs affected; genetic studies are underway

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Purebred, large-breed dogs
- German shepherd dog is the most commonly affected breed
- Other breeds include Pembroke and Cardigan Welsh corgi, Chesapeake Bay retriever, Rhodesian ridgeback, Irish setter, boxer, collie, standard poodle

Mean Age and Range

- Mean—9 years of age
- Range—older than 5 years of age
- Younger dogs also affected

Predominant Sex

- Males and females equally affected

SIGNS/OBSERVED CHANGES in the ANIMAL

- Subtle (insidious), progressive asymmetric wobbly, incoordinated or “drunken” appearing gait (known as “ataxia”)
- Hind-limb weakness or partial paralysis (paraparesis) leading to paralysis, eventually progressing to weakness or partial paralysis of all four limbs (known as “tetraparesis”) or paralysis of all four limbs (known as “tetraplegia”)
- Gait will show long-strided, spastic weakness or partial paralysis (paraparesis)
- Loss of proprioception early in disease course; “proprioception” is the normal subconscious awareness of the location of the limbs and movement
- Spinal reflexes usually present or exaggerated
- Decrease or absence of “knee-jerk” reflex (known as the “patellar reflex”) may be seen
- Loss of muscle mass due to decreased use (known as “disuse muscle atrophy”)
- Control of urination and defecation is lost late in disease course
- Lack of sensitivity to touch or pain along the spine (known as “paraspinal hyperesthesia”) is a key clinical feature
- Sensory perception remains unaffected

CAUSES

- Unknown (so called “idiopathic disease”)
- Hypothesized causes include immune-mediated disease, metabolic deficiencies, toxic-related disorder, oxidative stress and genetic disorder

RISK FACTORS

- Unknown



TREATMENT

HEALTH CARE

- Supportive care
- Breeds of small size may survive longer with degenerative myelopathy because the pet owner is able to give appropriate care more easily
- When the dog is unable to walk (known as being “nonambulatory”), s/he should be kept on a well-padded surface to prevent “bed sores” (known as “decubitus ulcers”) over bony prominences
- Keep hair trimmed, and skin clean and dry to prevent skin lesions (known as “urine scald”) that develop due to contact with urine, when the hair and skin remain damp, secondary to inability to control urination (known as “incontinence”)
- Physical therapy using range-of-motion and isometric exercises may help maintain limb mobility and muscle strength

ACTIVITY

- Exercise is encouraged to slow loss of muscle mass (disuse atrophy) of hind limbs
- Water-based physical therapy (known as “hydrotherapy”) can involve use of an under-water treadmill set up
- A wheel cart may assist with patient mobility

DIET

- Maintain a balanced diet
- Prevent weight gain

SURGERY

- None



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No drug has been proven to be effective in slowing or halting disease progression
- Some medications are being tried, including aminocaproic acid, vitamin E, or vitamin B₁₂



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat nervous system examinations
- Monitor urination
- Urinalysis and urine culture to check for urinary tract infection

PREVENTIONS AND AVOIDANCE

- Use of a well-padded surface to prevent “bed sores” (decubitus ulcers)
- Ensure that animal is urinating to prevent urine retention and urinary tract infection
- Keep hair trimmed, and skin clean and dry to prevent skin lesions (urine scald) that develop due to contact with urine, when the hair and skin remain damp, secondary to inability to control urination (incontinence)
- Prevent weight gain

POSSIBLE COMPLICATIONS

- Urine retention may increase the likelihood of urinary tract infections
- Local skin infections from “bed sores” (decubitus ulcers)

EXPECTED COURSE AND PROGNOSIS

- Paralysis of the hind limbs (known as “paraplegia”) occurs within 6 to 9 months from time of diagnosis
- Weakness or partial paralysis of all four limbs (tetraparesis) may be evident within 1 to 2 years from time of diagnosis
- Long-term prognosis is poor



KEY POINTS

- “Degenerative myelopathy” is a disease of the spinal cord that causes progressive weakness of the hind limbs (paraparesis)
- Long-term prognosis is poor
- Meticulous nursing care is crucial to preventing secondary complications in a recumbent patient

FIBROCARILAGINOUS EMBOLIC MYELOPATHY

(SPINAL CORD DISORDER CAUSED BY BLOCKAGE OF A BLOOD VESSEL)



BASICS

OVERVIEW

- “Fibrocartilaginous” refers to fibrocartilage; “fibrocartilage” is cartilage that contains bundles of collagen fibers; “cartilage” is a tough connective tissue found in joints and other body parts; “embolic” refers to an embolus or embolism; “embolus” is a material (such as a blood clot or foreign material) that blocks a blood vessel; “myelopathy” is a disorder of the spinal cord
- Fibrocartilaginous embolic myelopathy is a condition in which a piece of fibrocartilage breaks off the intervertebral disk and travels in the blood vessel until it blocks blood flow to the spinal cord
- Sudden (acute) death of spinal cord tissue due to lack of blood flow (known as “ischemic necrosis of the spinal cord”) caused by fibrocartilaginous emboli
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Giant- and large-breed dogs—highest number of cases
- Miniature schnauzers and Shetland sheepdogs—reported to have fibrocartilaginous embolic myelopathy at higher than anticipated rates; high levels of lipoprotein (compounds containing lipid [a group of compounds that contain fats or oils] and protein) in the blood (condition known as “hyperlipoproteinemia”) and resultant increased protein in the blood leading to sludging of the blood [known as “hyperviscosity”] are common in these breeds; hyperlipoproteinemia and hyperviscosity may contribute to sudden lack of blood supply that leads to death of tissues (known as “infarction”) of the spinal cord, without the presence of a piece of fibrocartilage material within the blood vessel (fibrocartilage embolus)

Mean Age and Range

- Most patients are 3 to 5 years of age
- Range, 16 weeks to 10 years

Predominant Sex

- Slight male predominance

SIGNS/OBSERVED CHANGES in the ANIMAL

- Mild trauma or vigorous exercise at the onset of signs is common
- Sudden (acute) onset
- Affected dog typically cries in pain; pain subsides in minutes to hours (at most)
- Signs of weakness or partial paralysis (known as “paresis”) or paralysis develop over a matter of seconds, minutes, or hours
- Condition stabilizes within 12 to 24 hours
- Nervous system deficits—usually involve one side primarily; unaffected side usually mildly affected or normal
- Pain—at onset of signs and then generally absent; usually subsides by the time the patient is being examined; may be felt for a few hours in severely affected patients
- Spinal cord injury—any level of the spinal cord can be affected, depending on the location of the piece of fibrocartilage material within the blood vessel (fibrocartilage embolus)
- Mild wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)
- Upper or lower motor neuron disease and related nervous system deficits; disease involving the nerve cells of the brain, brain stem and/or spinal cord that control the muscles is known as “upper motor neuron disease;” disease of the nerves that connect the spinal cord and muscles is known as “lower motor neuron disease”
- If signs progress beyond 24 hours, other diseases should be considered

CAUSES

- Unknown

RISK FACTORS

- Vigorous exercise may trigger the incident
- High levels of lipoprotein (compounds containing lipid [a group of compounds that contain fats or oils] and protein) in the blood (hyperlipoproteinemia)



TREATMENT

HEALTH CARE

- Inpatient—for immediate medical treatment and diagnostic procedures
- Keep recumbent patients on a padded surface; turn frequently to prevent pressure sores
- Assist and encourage patients to walk as soon as possible

ACTIVITY

- Restrict, until diagnosis is made

DIET

- Normal



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Methylprednisolone—may be beneficial, if given within the first 8 hours after onset of signs



FOLLOW-UP CARE

PATIENT MONITORING

- Sequential nervous system evaluations—during the first 12 to 24 hours after initial physical examination
- Nervous system status—2, 3, and 4 weeks after onset of clinical signs
- Lack of control of urination (known as “urinary incontinence”)—urinalysis and bacterial culture and sensitivity to detect urinary tract infection

PREVENTIONS AND AVOIDANCE

- Recurrence highly unlikely
- No known method of prevention

POSSIBLE COMPLICATIONS

- Inability to control bowel movements (known as “fecal incontinence”) and to control urine (urinary incontinence)
- Urinary tract infection
- Pressure sores and skin lesions that develop due to contact with urine, when the hair and skin remain damp (known as “urine scald”)
- Destruction of spinal cord tissue
- Euthanasia

EXPECTED COURSE AND PROGNOSIS

- Pain perception and upper motor neuron (involving the nerve cells of the spinal cord that control the muscles) signs—prognosis for

marked improvement generally good

- Loss of pain perception—prognosis poor
- Lack of reflexes (known as “areflexia”) of legs or sphincters—almost no chance of recovery
- Reduced purposeful movements and reflexes—functional recovery common; some degree of permanent deficit likely
- Progression of clinical signs from upper motor neuron (involving the nerve cells of the spinal cord that control the muscles) to lower motor neuron (involving the nerves that connect the spinal cord and muscles) and an enlarging area of sensory loss indicate development of a condition in which the motor neurons (nerve cells that control muscles) are destroyed, leading to progressive spinal cord disease that is not reversible (condition known as “myelomalacia”) and a hopeless prognosis; consider euthanasia
- Nervous system status—usually little change in the first 14 days after onset; most improvement occurs between days 21 and 42; replacement of myelin (a white material that covers certain nerve fibers; process known as “remyelination”) is complete in most patients within 6 to 12 weeks after onset; if no improvement after 21 to 30 days, recovery is highly unlikely



KEY POINTS

- Recovery from weakness or partial paralysis (paresis) or paralysis is slow and gradual, when it occurs
- Most patients need considerable supportive care at home during recovery

NAIL AND NAIL-BED DISORDERS



BASICS

OVERVIEW

- Nail and nail-bed disorders are a group of abnormalities or diseases that affect the nail or claw and/or the tissues surrounding the nail or claw (that is, the “nail bed”)
- “Onycho-“ or “onych-“ refers to the nail or claw
- Inflammation of soft tissue around the nail or claw (known as “paronychia”)
- Fungal infection of the nail or claw (known as “onychomycosis”)
- Brittle nails or claws that tend to split or break (known as “onychorrhexis”)
- Sloughing of the nail or claw (known as “onychomadesis”)
- Deformity of the nail or claw caused by abnormal growth (known as “nail dystrophy”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Dachshund—susceptible to brittle nails that tend to split or break (onychorrhexis)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Licking
- Lameness
- Pain
- Swelling, redness of the skin and tissues (known as “erythema”), and discharge from the area where the skin and nail or claw come together at the top of the toe (known as the “nail fold” or “claw fold”)
- Deformity or sloughing of one or more nails or claws

CAUSES

Inflammation of Soft Tissue Around the Nail or Claw (Paronychia)

- Infection or infectious disease—bacteria, fungus (known as “dermatophytosis”), yeast (*Candida*), demodectic mange mites (condition known as “demodicosis”), leishmaniasis
- Immune-mediated disease—types include the following: pemphigus, bullous pemphigoid, systemic lupus erythematosus, drug eruption, lupoid onychodystrophy
- Tumor or cancer—types include the following: squamous cell carcinoma, melanoma, eccrine carcinoma, osteosarcoma, subungual keratoacanthoma, inverted squamous papilloma
- Abnormal communication between an artery and a vein (known as an “arteriovenous fistula”)

Fungal Infection of the Nail or Claw (Onychomycosis)

- Dogs—*Trichophyton mentagrophytes* (usually generalized fungal infection of the skin, involving the nails)
- Cats—*Microsporum canis*

Brittle Nails or Claws that Tend to Split or Break (Onychorrhexis)

- Unknown cause (so called “idiopathic disease”)—especially in dachshunds; involves multiple nails
- Trauma
- Infection—fungal infection (dermatophytosis), leishmaniasis

Sloughing of the Nail or Claw (Onychomadesis)

- Trauma
- Infection
- Immune-mediated disease—types include the following: pemphigus, bullous pemphigoid, systemic lupus erythematosus, drug eruption, lupoid onychodystrophy
- Decreased or inadequate blood flow (known as “vascular insufficiency”)—inflammation of the blood vessels (known as “vasculitis”); clumping together or agglutination of red-blood cells when the temperature of the cells drops below normal body temperature (known as “cold agglutinin disease”), such as exposure of the legs to cold weather

- Tumor or cancer—types include the following: squamous cell carcinoma, melanoma, eccrine carcinoma, osteosarcoma, subungual keratoacanthoma, inverted squamous papilloma
- Unknown cause (so called “idiopathic disease”)

Deformity of the Nail or Claw Caused by Abnormal Growth (Nail Dystrophy)

- Condition caused by excessive levels of growth hormone, leading to enlargement of bone and soft-tissues in the body (known as “acromegaly”)
- Increased levels of thyroid hormone in the cat (known as “feline hyperthyroidism”)
- Zinc-responsive skin disorder (known as “zinc-responsive dermatosis”)
- Congenital (present at birth) malformations of the nail or claw

RISK FACTORS

- Inflammation of soft tissue around the nail or claw (paronychia) due to infectious causes—decreased ability to develop a normal immune response (known as “immunosuppression”), which may be related to immune-system problem of the body or to the use of medications to decrease the immune response; feline leukemia virus (FeLV) infection; trauma; and diabetes mellitus (sugar diabetes)
- Sloughing of the nail or claw due to bacterial infection (bacterial onychomadesis)—excessively short nail trimming (into the quick) postulated to increase likelihood of bacterial infection and subsequent sloughing of the nail or claw



TREATMENT

HEALTH CARE

Inflammation of Soft Tissue Around the Nail or Claw (Paronychia)

- Surgical removal of the hard part of the nail (known as the “nail plate” or “shell”)—provide adequate drainage; bandage foot following procedure
- Antimicrobial soaks
- Identify underlying condition and treat specifically

Fungal Infection of the Nail or Claw (Onychomycosis)

- Antifungal soaks—chlorhexidine, povidone iodine, lime sulfur
- Surgical removal of the hard part of the nail (nail plate or shell)—may improve response to medication administered by mouth or injection (known as “systemic medication”)
- Amputation of the third bone of the toe (known as the “third phalanx”), which is continued by the nail or claw

Brittle Nails or Claws that Tend to Split or Break (Onychorrhexis)

- Repair with fingernail glue (type used to attach false nails in humans), as performed or directed by your pet’s veterinarian
- Remove splintered pieces
- Amputation of the third bone of the toe (third phalanx), which is continued by the nail or claw
- Treat underlying cause

Sloughing of the Nail or Claw (Onychomadesis)

- Antimicrobial soaks
- Treat underlying cause

Deformity of the Nail or Claw Caused by Abnormal Growth (Nail Dystrophy)

- Treat underlying cause

Tumor or Cancer

- Depends on biologic behavior of specific tumor
- Surgical removal of the tumor
- Amputation of toe
- Amputation of leg
- Chemotherapy
- Radiation therapy



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Bacterial infection/inflammation of soft tissue around the nail or claw (paronychia)—antibiotics based on bacterial culture and sensitivity, administered by mouth or injection (systemic antibiotics); cephalosporins pending culture result
- Yeast (*Candida*) infection/inflammation of soft tissue around the nail or claw (paronychia)—ketoconazole administered by mouth (systemic treatment); nystatin or miconazole applied to the affected area directly (topical treatment)
- Fungal infection of the nail or claw (onychomycosis)—griseofulvin or ketoconazole administered by mouth (systemic treatment) for 6 to 12 months until negative fungal cultures; itraconazole administered by mouth for 3 weeks and then as directed by your pet's veterinarian
- Sloughing of the nail or claw (onychomadesis)—depends on cause; medication to decrease the immune response (known as “immunosuppressive therapy”) for immune-mediated diseases
- Often-used medications include cyclosporine, tetracycline with niacinamide, pentoxifylline, vitamin E, essential fatty acid supplementations, and chemotherapeutic agents (such as azathioprine, chlorambucil)



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on underlying cause

PREVENTIONS AND AVOIDANCE

- Depend on underlying cause

POSSIBLE COMPLICATIONS

- Depend on underlying cause

EXPECTED COURSE AND PROGNOSIS

- Bacterial infection/inflammation of soft tissue around the nail or claw (paronychia) or fungal infection of the nail or claw (onychomycosis)—treatment may be prolonged and response may be influenced by underlying factors that decrease the immune response (immunosuppressive factors)
- Fungal infection of the nail or claw (onychomycosis) and brittle nails or claws that tend to split or break (onychorrhexis)—may require amputation of the third bone of the toe (third phalanx), which is continued by the nail or claw, in order to get resolution
- Deformity of the nail or claw caused by abnormal growth (nail dystrophy)—prognosis is good when underlying cause can be effectively treated (such as cases with increased levels of thyroid hormone [hyperthyroidism] or zinc-responsive skin disorders [dermatosis])
- Sloughing of the nail or claw (onychomadesis)—prognosis depends on underlying cause; immune-mediated diseases and blood vessel/blood flow (vascular) problems carry a more guarded prognosis than do trauma or infectious causes
- Tumor or cancer—some can be totally removed surgically or removed by amputation of the toe; others are highly malignant and may have already spread (known as “metastasis”) by the time of diagnosis



KEY POINTS

- Nail and nail-bed disorders are a group of abnormalities or diseases that affect the nail or claw and/or the tissues surrounding the nail or claw (that is, the “nail bed”)

NASAL DISCHARGE



BASICS

OVERVIEW

- “Nasal discharge” is discharge from the nose; it may be clear, blood tinged or may contain mucus and/or pus; it also may be from bleeding in the nose and nasal passages (known as “epistaxis” or a “nosebleed”) or may contain food debris
- “Sneezing” is the forceful expelling of air through the mouth and nose, usually caused by irritation of the lining of the nose and nasal passages

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Hunting dogs—foreign body
- Dogs with a long head and nose (known as “dolichocephalic dogs,” such as the collie or Afghan hound)—aspergillosis, a fungal infection

Mean Age and Range

- Young animals—cleft palate; nasal polyp; disorder in which the normal secretion clearance mechanism is defective (known as “ciliary dyskinesia”); decreased ability to produce immunoglobulins, immune proteins or antibodies necessary for fighting disease (known as an “immunoglobulin deficiency”)
- Older animals—tumors of the nose and nasal passages; primary dental disease (tooth-root abscess)

Predominant Sex

- Male dogs may have a higher incidence of fungal infection of the nose than do females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sneezing—often accompanies discharge from the nose
- Discharge may be clear, blood tinged or may contain mucus and/or pus; it also may be from bleeding in the nose and nasal passages (epistaxis or a nosebleed) or may contain food debris; it is important to observe both the initial and current character of the discharge, as well as whether it started originally from one nostril (known as “unilateral discharge”) or both nostrils (known as “bilateral discharge”)
- Noisy breathing when inhaling (known as “stertor”)—noisy breathing frequently noted, especially when the animal is sleeping
- Response to previous antibiotic therapy common, due to secondary bacterial infection
- Discharge or dried discharge on the hair of the muzzle or forelimbs
- May note decreased air flow through the nose, particularly with tumors of the nose or nasal passages
- Dental disease
- Bony involvement—with a tumor or fourth premolar tooth abscess; may be detected as swelling of the face or hard palate (roof of the mouth) or as pain secondary to fungal or bacterial infection/inflammation of the bone marrow and bone (known as “osteomyelitis”) or cancer
- Loss of pigment in the lining of the nose (known as “mucosal depigmentation”)—often observed with aspergillosis, a fungal infection
- Enlargement of the lymph nodes under the lower jaw (known as “mandibular lymphadenomegaly”)—cancer, fungal infection, dental disease
- Polyp—may be visible on examination of the ear, or noted during examination of the mouth as pushing on the soft palate (the soft portion of the roof of the mouth, located between the hard palate and the throat)
- Inflammation of the choroid and retina (known as “chorioretinitis”)—may be seen with canine distemper virus infection or cryptococcosis, a fungal infection; the choroid is located immediately under the retina and is part of the middle-layer of the eyeball that contains the blood vessels; the retina contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision

CAUSES

- Discharge from one nostril (unilateral discharge)—often associated with local problems (that is, in or near the nose or nasal passages) rather than generalized (systemic) disease; may include foreign body in the nose or nasal passages; dental-related disease; fungal infections; tumors of the nose or nasal passages; facial nerve damage leading to extreme dryness of the lining of the nose and nasal passages (known as “xeromycteria”)
- Discharge from both nostrils (bilateral discharge)—infectious agents (such as feline herpes virus [cause of feline viral rhinotracheitis])

or calicivirus, canine distemper virus, secondary bacterial infection); immunoglobulin A (IgA) deficiency (immunoglobulin A (IgA) is an immune protein, found in the lining of the moist tissues of the body; it functions as a protective barrier to prevent or limit antigens [substance to which the immune system is responding and producing antibodies] and disease-causing microorganisms from entering the body through these tissues); airborne irritant; allergy; disorder in which the normal secretion clearance mechanism is defective (ciliary dyskinesia); inflammation of the nose (known as “rhinitis”)

- Discharge from one nostril (unilateral discharge) progressing to discharge from both nostrils (bilateral)—*Aspergillus* infection, a fungal infection; tumor of the nose or nasal passages

- Either discharge from one nostril (unilateral) or from both nostrils (bilateral)—nosebleed (epistaxis); foreign body; more generalized disease (that is, disease involving other body systems) causing signs of nasal discharge; parasites of the nose

- Generalized disease (that is, disease involving other body systems) causing signs of nasal discharge—long-term (chronic) pneumonia, long-term (chronic) vomiting

RISK FACTORS

- Dental disease
- Foreign bodies—outdoor animals
- Infectious disease—poorly vaccinated animal, kennel situations, exposure to other animals
- Nasal aspergillosis, a fungal infection—dogs bedded on straw
- Nasal mites (type of parasite)—kennel-raised dogs
- Suppression of immune response, as by drugs (known as “immunosuppression”); long-term (chronic) steroid use; and feline leukemia virus (FeLV) or feline immunodeficiency virus (FIV) infection
- Long-term (chronic), low grade pneumonia
- Long-term (chronic) vomiting
- Long-term (chronic) inflammation of the ear (known as “otitis”), leading to facial nerve damage



TREATMENT

HEALTH CARE

- Outpatient—except if surgery is required
- Adequate hydration, nutrition, warmth, and hygiene (keeping nostrils clean)—important with long-term (chronic) sneezing and nasal discharge
- Inpatient—for exploratory surgery of the nose (known as “rhinotomy”) or sinuses (known as “sinusotomy”); topical (directly applied into the nose) treatment for aspergillosis, a fungal infection

SURGERY

- Exploratory surgery of the nose (known as “rhinotomy”) or sinuses (known as “sinusotomy”)
- Removal of foreign body
- Removal of tumors
- Treatment of dental disease



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Secondary bacterial infection—antibiotics; use an antibiotic with good gram-positive spectrum of activity, such as amoxicillin, amoxicillin-clavulanic acid (Clavamox®), clindamycin, azithromycin (Zithromax®), cephalosporins
- Attempt to dry up nasal secretions—decongestants (ephedrine); medications applied to the nasal passages directly to shrink blood vessels (known as “topical vasoconstrictors,” such as Neosynephrine® or oxymetazoline), as directed by your pet’s veterinarian
- Dental-associated inflammation of the nose (rhinitis)—antibiotics; dental surgery as indicated
- Foreign body in the nose—removal of foreign body, followed by antibiotics
- Parasites in the nose or nasal passages—ivermectin or milbemycin (as directed by your pet’s veterinarian) to treat *Pneumonyssoides*; fenbendazole to treat *Capillaria*

- Nonspecific inflammation of the nose and nasal passages—prednisolone or piroxicam to decrease inflammation
- Canine nasal aspergillosis, a fungal infection—enilconazole or clotrimazole directly applied into the nose (topical treatment)
- Feline cryptococcosis or sporotrichosis (fungal infections)—itraconazole or fluconazole administered by mouth
- Cancer—radiation therapy and chemotherapy
- Extreme dryness of the lining of the nose and nasal passages (xeromycteria)—steroids and antibiotics; pilocarpine in attempt to stimulate secretions



FOLLOW-UP CARE

PATIENT MONITORING

- Nasal discharge and sneezing—note changes in frequency, volume, and character
- Repeat evaluation of the nose by using a special lighted instrument called an “endoscope” (procedure is “rhinoscopy”)—indicated to ensure adequate response to treatment of fungal inflammation of the nose (rhinitis)
- Recheck chest X-rays or evaluate the lower airways by using a special lighted instrument (endoscope; procedure known as “bronchoscopy”)—monitor response to treatment for long-term (chronic) pneumonia

PREVENTIONS AND AVOIDANCE

- Vaccinate animals against diseases that cause nasal discharge (such as canine distemper virus and feline calicivirus)

POSSIBLE COMPLICATIONS

- Loss of appetite—especially in cats
- Extension of primary disease (for example, fungal infection or tumor) into the mouth, eye, or brain
- Breathing distress—with blockage or obstruction of the nose and/or nasal passages
- Involvement of the cribriform plate (bony plate located between the nasal passages and the brain) in dogs with aspergillosis, a fungal infection—central nervous system (brain) damage during treatment with anti-fungal medications applied into the nasal passages directly

EXPECTED COURSE AND PROGNOSIS

- Depend on cause



KEY POINTS

- Nasal discharge” is discharge from the nose; it may be clear, blood tinged or may contain mucus and/or pus; it also may be from bleeding in the nose and nasal passages (epistaxis or a nosebleed) or may contain food debris
- Adequate hydration, nutrition, warmth, and hygiene (keeping nostrils clean)—important with long-term (chronic) sneezing and nasal discharge
- Vaccinate animals against diseases that cause nasal discharge (such as canine distemper virus and feline calicivirus)

KIDNEY STONES (NEPHROLITHIASIS)



BASICS

OVERVIEW

- “Nephrolithiasis” is the medical term for the presence of stones in the kidneys; “nephrolith” is the medical term for a kidney stone; “urolith” is the general term for urinary tract stones (that is, stones located anywhere in the urinary tract)
- The kidney is composed of thousands of “nephrons” (the functional units of the kidney, each consisting of the “glomerulus” [a tuft of blood capillaries—the “blood filter”] and a series of tubes, through which the filtered fluid flows, as urine is produced; the tubes drain into “collecting ducts” through which urine flows; the collecting ducts join together and eventually enter the renal pelvis; the “renal pelvis” is a funnel-shaped structure through which urine flows into the ureter (the tube between the kidney and bladder)
- Kidney stones (nephroliths) are either aggregates of crystals or stones located in the renal pelvis or collecting diverticula of the kidney
- The most common minerals found in the stones of the urinary tract (uroliths) are used to name the particular stone, such as “calcium oxalate uroliths,” “struvite uroliths,” and “urate uroliths”
- Kidney stones (nephroliths) or fragments of kidney stones may pass into the ureters; the “ureters” are the tubes from the kidneys to the bladder
- Kidney stones (nephroliths) that are not infected, not causing blockage or obstruction of urine flow or clinical signs, and not progressively enlarging are called “inactive kidney stones”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

Canine

- Kidney stones containing calcium and oxalate or oxalic acid (known as “calcium oxalate nephroliths”)—miniature schnauzer, Lhasa apso, Yorkshire terrier, miniature poodle, and shih tzu
- Kidney stones containing magnesium ammonium phosphate (known as “struvite;” stones are known as “struvite nephroliths”)—miniature schnauzer, bichon frise, shih tzu, Yorkshire terrier, Lhasa apso, cocker spaniel, and miniature poodle
- Kidney stones containing uric acid or urate (known as “urate nephroliths”)—Dalmatian, Yorkshire terrier, and English bulldog
- Kidney stones containing cystine (known as “cystine nephroliths”)—Newfoundland

Feline

- Breeds reported to have kidney stones (nephroliths) include the following: domestic shorthair (33%), domestic longhair (17%), Persian (8%), Siamese (6%)

Mean Age and Range

- Dogs—mean age of affected animals, 9 years (range, 4 months to 14 years of age)
- Cats—mean age of affected animals, 8 years (range, 2 months to 18 years of age)

Predominant Sex

- Overall, kidney stones (nephroliths) in dogs are slightly more common in females than in males; females are more likely than males to develop struvite kidney stones; males are more likely than females to develop calcium oxalate, cystine, and urate kidney stones
- In cats, kidney stones (nephroliths) are slightly more common in females than in males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Many patients have no clinical signs, and the kidney stones (nephroliths) are found during the diagnostic workup of other medical problems
- Blood in the urine (known as “hematuria”), vomiting, and recurrent urinary tract infection; difficulty or painful urination (known as “dysuria”) and frequent voiding of small volumes (known as “pollakiuria”) in animals with urinary tract infection
- Signs attributable to excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) in animals with blockage of urine flow involving both ureters or kidneys or kidney failure; possible signs include lack of appetite (known as “anorexia”), sluggishness (lethargy), vomiting, and ulcers in the mouth
- Signs referable to stones (uroliths) in the lower urinary tract (the bladder and urethra; the “urethra” is the tube from the bladder to the outside, through which urine flows out of the body), if stones are present in the upper urinary tract (kidneys and ureters) and the lower urinary tract; possible signs include difficulty or painful urination (dysuria), frequent voiding of small volumes (pollakiuria), straining to urinate (known as “stranguria”), and reduced or absent flow of urine
- So-called “renal colic” with sudden (acute) abdominal/ lumbar pain and vomiting is uncommon

CAUSES

- Over saturation of the urine with stone-forming minerals may contribute to development of urinary tract stones (uroliths)
- Urinary tract stones containing calcium and oxalate or oxalic acid (calcium oxalate uroliths)—increased levels of calcium in the urine (known as “hypercalciuria”), increased levels of calcium in the blood (known as “hypercalcemia”), low levels of citrate in the urine (known as “hypocitraturia”), increased levels of oxalate or oxalic acid in the urine (known as “hyperoxaluria”), abnormality in calcium and phosphorus regulation in the body (known as “hyperparathyroidism”), excessive calcium intake in the diet
- Urinary tract stones containing calcium and phosphate (known as “calcium phosphate uroliths”)—long-term (chronic) bleeding in the kidneys (cats), increased levels of calcium in the blood (hypercalcemia), abnormality in calcium and phosphorus regulation in the body (hyperparathyroidism), excessive calcium and phosphorus in the diet
- Urinary tract stones containing magnesium ammonium phosphate or struvite (struvite uroliths)—urinary tract infection, diets that produce high (alkaline) urine pH
- Urinary tract stones containing uric acid or urate (urate uroliths)—inherited disorder (Dalmatians), portosystemic shunt (condition in which blood vessels allow blood to flow abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)
- Urinary tract stones containing xanthine (known as “xanthine uroliths”)—allopurinol (a medication used to reduce uric acid) administration and high dietary purine intake in dogs susceptible to developing urinary tract stones containing uric acid or urate (urate uroliths)
- Urinary tract stones containing cystine (cystine uroliths)—increased levels of cystine in the urine (known as “cystinuria”)

RISK FACTORS

- Alkaline urine (urine with high pH)—struvite and calcium phosphate uroliths
- Acid urine (urine with low pH)—calcium oxalate, cystine, urate, and xanthine uroliths
- Urine retention and formation of highly concentrated urine
- Lower urinary tract infection—infection may move up from the lower urinary tract to the kidneys and result in bacterial infection/inflammation of the kidney (known as “pyelonephritis”)
- Conditions that increase the likelihood of developing urinary tract infection (such as surgical removal of the penis with creation of a new opening into the urethra [known as “perineal urethrostomy”]; abnormalities during development of the urinary tract in which the ureters [tubes from the kidneys to the bladder] may not attach to the bladder properly or may attach to reproductive organs—when this occurs, they are called “ectopic ureters” and one or both can terminate in the distal urethra, uterus, or vagina; increased levels of steroids produced by the adrenal glands [known as “hyperadrenocorticism” or “Cushing’s disease”]; reflux of urine from the bladder back into the ureter(s); and administration of steroid-containing medications



TREATMENT

HEALTH CARE

- Manage patients with inactive kidney stones (nephroliths) as outpatients
- Medical protocols to dissolve stones can be administered to outpatients
- Removal of kidney stones (nephroliths) by surgery or extracorporeal shock wave lithotripsy requires hospitalization; extracorporeal shock wave lithotripsy is a medical procedure in which the stone is broken up within the urinary tract using some type of energy or sound wave

ACTIVITY

- Unlimited

DIET

- Medical protocols to dissolve kidney stones (nephroliths) requires a diet appropriate to the specific chemical make-up of the stone

SURGERY

- Indications for removal of kidney stones (nephroliths)—blockage or obstruction of urine flow; recurrent infection; kidney stones are causing signs of disease (known as “symptomatic nephroliths”); progressive enlargement of the kidney stone; and a nonfunctional kidney on the opposite side of the kidney containing a stone
- Treatment options for kidney stones (nephroliths)—dissolve the stone through medical treatment, surgery, and extracorporeal shock wave lithotripsy, a medical procedure in which the stone is broken up within the urinary tract using some type of energy or sound wave
- Stones in the ureters (known as “ureteroliths”) or in the kidney (nephroliths) causing complete blockage or obstruction of urine flow are not responsive to being dissolved through medical treatment, thus surgery is necessary
- Extracorporeal shock wave lithotripsy—safe and effective method of treating canine kidney stones (nephroliths) and stones in the

ureters (ureteroliths); stone fragments pass down the ureter into the bladder and are voided in the urine

- Extracorporeal shock wave lithotripsy—not as effective for treatment of kidney stones (nephroliths) and stones in the ureters (ureteroliths) in cats compared with effectiveness in dogs



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics selected on the basis of urine bacterial culture and sensitivity testing, as necessary
- Antibiotics are recommended at the time of the procedure when infected kidney stones (nephroliths) are treated by extracorporeal shock wave lithotripsy, a medical procedure in which the stone is broken up within the urinary tract using some type of energy or sound wave or are removed surgically
- Medical protocols to dissolve stones are limited to struvite, urate, and cystine stones (uroliths)
- Medical protocols to dissolve *struvite* kidney stones (nephroliths) include a diet to dissolve the stones (Hill's Prescription Diet® s/d®) and appropriate antibiotic therapy (if patient has a urinary tract infection) for the duration of treatment
- Medical protocols to dissolve canine *urate* kidney stones (nephroliths) can be attempted by a protein- and purine-restricted, alkalinizing diet (Hill's Prescription Diet® Canine u/d®), allopurinol, and supplemental potassium citrate, as needed to maintain urine pH at approximately 7.0
- Medical protocols to dissolve canine *cystine* kidney stones (nephroliths) can be attempted using a protein-restricted, alkalinizing diet (Hill's Prescription Diet® Canine u/d®), N-(2-mercaptopropionyl) glycine (2-MPG or THIOLA®), and supplemental potassium citrate, as needed to maintain urine pH at approximately 7.5
- Kidney stones containing calcium and oxalate or oxalic acid (calcium oxalate nephroliths), the most common mineral composition in dogs and cats, are not responsive to being dissolved through medical treatment



FOLLOW-UP CARE

PATIENT MONITORING

- Abdominal X-rays or ultrasound examinations, urinalysis, and urine culture every 3 to 6 months to detect recurrence of kidney stones (nephroliths)
- Dogs treated with extracorporeal shock wave lithotripsy, a medical procedure in which the stone is broken up within the urinary tract using some type of energy or sound wave—check every 2 to 4 weeks by X-rays and ultrasound examination until fragments of the kidney stone have passed through the lower urinary tract (bladder and urethra)

PREVENTIONS AND AVOIDANCE

- Eliminate factors that increase the likelihood of developing a particular type of urinary tract stone, increase urine volume (thus decreasing the concentration of stone-causing minerals), and correct factors contributing to urine retention

POSSIBLE COMPLICATIONS

- Blockage or obstruction of urine flow
- Dilation of the funnel-shaped structure (renal pelvis) through which urine flows into the ureter due to blockage of urine flow (known as “hydronephrosis”)
- Kidney failure
- Recurrent urinary tract infection
- Bacterial infection/inflammation of the kidney (pyelonephritis)

EXPECTED COURSE AND PROGNOSIS

- Highly variable; depends on type, location, and size of the kidney stone, and the presence of secondary complications (such as blockage or obstruction of urine flow, kidney failure, urinary tract infection)
- Inactive nephroliths may remain inactive for years, resulting in an excellent prognosis
- Extracorporeal shock wave lithotripsy (a medical procedure in which the stone is broken up within the urinary tract using some type of energy or sound wave) to treat dogs with kidney stones (nephroliths)—return to normal health and excellent prognosis
- Prognosis for patients with kidney failure caused by the presence of stones in the kidney (nephrolithiasis) depends on the severity and rate of progression of kidney failure



KEY POINTS

- Inactive kidney stones (nephroliths)—may not require removal, but should be monitored periodically by urinalysis, urine culture, and X-rays; potentially can cause blockage or obstruction of urine flow at any time, which can result in dilation of the funnel-shaped structure (renal pelvis) through which urine flows into the ureter due to blockage of urine flow (hydronephrosis) without clinical signs, so conservative management and monitoring carries a slight risk of undetected and potentially irreversible kidney damage, which must be weighed against the potential kidney damage from kidney surgery to remove the stone
- Kidney stones (nephroliths) tend to recur after removal; monitor the patient every 3 to 6 months

NEPHROTIC SYNDROME



BASICS

OVERVIEW

- “Nephrotic” is an adjective that refers to anything that relates to disease of the kidney
- The kidney filters the blood and removes various waste products from the body as it produces urine; the kidney also is involved in maintaining the normal fluid volume of the body; each kidney is composed of thousands of nephrons (the functional units of the kidney, each consisting of the glomerulus [a tuft of blood capillaries—the “blood filter”] and a series of tubes and ducts, through which the filtered fluid flows, as urine is produced)
- Nephrotic syndrome is a medical condition in which the animal has significant levels of protein in its urine (known as “proteinuria”); low levels of albumin [a type of protein] in its blood (known as “hypoalbuminemia”); high levels of cholesterol in its blood (known as “hypercholesterolemia”); and fluid accumulation in the abdomen (known as “ascites”), in the space between the chest wall and the lungs (known as “pleural effusion”), and/or under the skin (known as “subcutaneous edema”)
- Nephrotic syndrome occurs secondary to glomerular disease (such as glomerulonephritis, kidney or renal amyloidosis)
- Glomerulonephritis is inflammation and accompanying dysfunction of glomeruli (plural of glomerulus); most commonly due to the presence of immune complexes in the glomerulus
- Renal amyloidosis is a condition in which insoluble proteins (amyloid) are deposited outside the cells of the kidneys, compromising the normal function of kidney

GENETICS

- No breed appears to be at increased risk of developing the nephrotic syndrome complication with glomerular disease
- Familial glomerular diseases (glomerular disease that runs in certain families of animals) have been reported in several breeds, including Bernese mountain dogs, bull terriers, Dalmatians, Samoyeds, Doberman pinschers, cocker spaniels, Newfoundlands, rottweilers, greyhounds, soft-coated wheaten terriers, and cats
- Familial amyloidosis (condition that runs in certain families of animals, in which insoluble proteins [amyloid] are deposited outside the cells in the kidney, compromising its normal function) is common in Chinese shar peis; sporadic cases of affected litters have been reported in other breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- No breed appears to be at increased risk of developing the nephrotic syndrome complication with glomerular disease

Mean Age and Range

- Mean age of dogs with glomerulonephritis—6.5 to 7.0 years; range, 0.8 to 17 years
- Cats with glomerulonephritis—mean age at presentation is 4.0 years
- Most dogs and cats with kidney or renal amyloidosis (in which insoluble proteins [amyloid] are deposited outside the cells in the kidney, compromising its normal function) are over 5 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Fluid build-up under the skin that retains an indentation produced by finger pressure on the tissue (known as “pitting subcutaneous edema”) and/or fluid build-up in the abdomen (ascites) are the most common reason the owner seeks veterinary care
- Occasionally, signs associated with an underlying infectious or inflammatory disease or cancer may be the primary reason why owners seek veterinary care
- Rarely, dogs may exhibit sudden (acute) difficulty breathing (known as “dyspnea”) or severe panting due to fluid build-up in the space between the chest wall and lungs (pleural effusion), fluid build-up in the lungs (known as “pulmonary edema”), or blood clots in the lungs (known as “pulmonary thromboembolism”)
- Complications of high blood pressure (hypertension): bleeding in the back of the eye (known as “retinal hemorrhage”) or loss of attachment of the retina to the underlying structures of the eye (known as “retinal detachment”); fluid build-up/swelling of the optic disk (known as “papilledema”); irregular heart beats (known as “arrhythmias”) and/or heart murmurs, secondary to enlargement of the left ventricle (known as “left ventricular hypertrophy”)
- Difficulty breathing (dyspnea) and bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”) in dogs with fluid build-up in the space between the chest wall and lungs (pleural effusion) or blood clots in the lungs (pulmonary thromboembolism)

CAUSES

- Nephrotic syndrome occurs secondary to glomerular disease (such as glomerulonephritis, kidney or renal amyloidosis)
- Glomerulonephritis or amyloidosis may occur secondary to chronic inflammation (such as caused by infection, cancer, and immune-mediated disease)



TREATMENT

HEALTH CARE

- Most patients can be treated as outpatients; exceptions include patients that have very high levels of urea and other nitrogenous waste products in the blood (condition known as “uremia” or “azotemia”) and/or high blood pressure (hypertension); patients with blood-clotting disease (known as “thromboembolic disease”)
- Tapping the abdomen to drain off excessive fluid (known as “abdominocentesis”) and/or tapping the chest to drain off excessive fluid (known as “thoracocentesis”) is used in patients with severe breathing difficulties (known as “respiratory distress”) and abdominal discomfort caused by fluid build-up in the abdomen (ascites) and/or fluid build-up in the space between the chest wall and the lungs (pleural effusion); in most patients, removal of fluid will only increase the rate of fluid accumulation and contributes to electrolyte abnormalities via removal of large amounts of sodium from the body
- Plasma transfusion is not indicated for treatment of low levels of albumin (a protein) in the blood (hypoalbuminemia); large amounts of plasma are required to significantly increase serum albumin concentrations, and transfused protein does not remain in the circulation for long
- Intravenous human albumin is only indicated in the very rare cases where patients develop life-threatening complications (such as fluid build-up in the lungs [pulmonary edema]; fluid build-up in the space between the chest wall and lungs [pleural effusion])

ACTIVITY

- Restriction of activity may be beneficial because of the possibility of blood-clotting disease (thromboembolic disease)

DIET

- Sodium-reduced, high-quality, low-quantity protein diets—commercial “kidney diets” meet these criteria
- Dietary protein supplementation formerly was recommended to offset the effects of loss of protein into the urine (proteinuria); however, normal or high dietary protein may contribute to the progression of kidney disease, thus dietary therapy should include a reduced (not restricted) amount of high-quality protein (such as found in Hill’s Prescription Diet® (canine or feline) k/d®)

SURGERY

- Biopsy is required to differentiate between glomerulonephritis and amyloidosis



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Fluid Build-Up (Edema and Ascites)

- Dietary sodium reduction
- Reserve tapping the abdomen to remove excessive fluid (abdominocentesis) and drugs to increase the amount of urine (fluid) eliminated from the body (known as “diuretics”) for patients with severe breathing difficulties (respiratory distress) and abdominal discomfort
- Overzealous use of diuretics may cause dehydration and sudden (acute) kidney decompensation

Protein in the Urine (Proteinuria)

- Angiotensin-converting enzyme (ACE) inhibitors decrease loss of protein into the urine (proteinuria); in one prospective study, enalapril (an ACE inhibitor) decreased high blood pressure and protein loss into the urine and decreased progression of kidney disease in dogs with glomerulonephritis of unknown cause (condition known as “idiopathic glomerulonephritis”)—proteinuria is directly toxic to kidney tubules; therefore, ACE-inhibitor therapy should be initiated at the time of diagnosis, unless the patients has very high levels of urea and other nitrogenous waste products in the blood (uremia or azotemia)



FOLLOW-UP CARE

PATIENT MONITORING

● Urinary protein:creatinine ratio; serum urea nitrogen, creatinine, albumin, and electrolyte concentrations; blood pressure; and body weight; ideally, recheck examinations should occur 1, 3, 6, 9, and 12 months after initiation of treatment

POSSIBLE COMPLICATIONS

- Long-term (chronic) kidney insufficiency or failure
- Blood clots to the lungs (pulmonary thromboembolism)

EXPECTED COURSE AND PROGNOSIS

- If the underlying cause cannot be identified and corrected, glomerulonephritis and amyloidosis usually are progressive, eventually resulting in chronic kidney failure
- Once azotemia (high levels of urea and other nitrogenous waste products in the blood) and kidney failure develop, prognosis is often poor due to rapidly progressive disease



KEY POINTS

- Nephrotic syndrome is a medical condition in which the animal has significant levels of protein in its urine (known as “proteinuria”); low levels of albumin [a type of protein] in its blood (known as “hypoalbuminemia”); high levels of cholesterol in its blood (known as “hypercholesterolemia”); and fluid accumulation in the abdomen (known as “ascites”), in the space between the chest wall and the lungs (known as “pleural effusion”), and/or under the skin (known as “subcutaneous edema”)
- Nephrotic syndrome occurs secondary to glomerular disease (such as glomerulonephritis, kidney or renal amyloidosis)
- If the underlying cause cannot be identified and corrected, glomerulonephritis and amyloidosis usually are progressive, eventually resulting in chronic kidney failure
- Biopsy is required to differentiate between glomerulonephritis and amyloidosis
- Once azotemia (high levels of urea and other nitrogenous waste products in the blood) and kidney failure develop, prognosis is often poor due to rapidly progressive disease

OBESITY



BASICS

OVERVIEW

- An excess of body fat, frequently resulting in adverse health effects
- Even a moderate excess in body fat can increase incidence of disease (known as “morbidity”) and reduce lifespan

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- All ages, with the greatest prevalence (nearly 50%) in middle-aged dogs and cats

Predominant Sex

- Most common in neutered, indoor pets

SIGNS/OBSERVED CHANGES in the ANIMAL

- Weight gain
- Exercise intolerance may be reported
- Excess body fat and high body condition score or “BCS” (estimate of weight status [under or overweight] as compared to normal weight)

CAUSES

- Obesity is caused by an imbalance between calorie or energy intake and calorie or energy expenditure, with intake exceeding expenditure
- Neutering, decreased opportunities for activity, and age can reduce expenditure of energy
- Overfeeding of high calorie foods, frequently alternating foods, and provision of excess treats contribute to excess calorie or energy intake
- Low levels of thyroid hormone (known as “hypothyroidism”), insulin-secreting tumor (known as an “insulinoma”), or high levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”) are infrequent causes of obesity



TREATMENT

HEALTH CARE

- Weight loss, induced by reducing calorie intake below calorie or energy expenditure
- Successful weight loss also requires long-term maintenance of the reduced weight
- Weight loss and maintenance of reduced weight depend on changes in the way the owner feeds and interacts with the pet
- The owner should assess and monitor the body condition score (BCS; estimate of weight status [under or overweight] as compared to normal weight) of his or her pet

ACTIVITY

- Calorie or energy restriction results in compensatory decreases in basal energy expenditure or metabolism; increased activity helps compensate for this decrease in metabolism and provides alternate opportunities for owner-pet interactions
- Leash walking for dogs and trained cats—at least 15 minutes twice daily
- Activities such as “fetch”, interactive toys for cats, or playing with a laser light
- Food balls—built to hold treats or kibbles and randomly release them while the dog or cat plays; food used in the ball must be included as part of the daily calorie allowance

DIET

- Get written instructions regarding specific amounts to provide, using the agreed upon reducing diet (a “cup” of food refers to an 8-oz measuring cup)
- Increased dietary protein facilitates loss of body fat, while minimizing loss of lean body mass (LBM), which is the metabolically active tissue—preserving LBM should help with long-term weight control by maintaining a higher resting energy requirement; protein also stimulates metabolism, increases energy expenditure, and contributes to the feeling of being full or satisfied (known as “satiety”)
- Dietary fiber provides little dietary energy, so it helps reduce total calories in the diet; fiber also stimulates intestinal metabolism and energy utilization, and contributes to the feeling of being full or satisfied (satiety)
- Fat is calorie or energy dense, so low-fat diets are lower in energy

- Calories should be restricted, without excessive restriction of essential nutrients; a low-calorie therapeutic diet with an increased nutrient-to-calorie ratio is recommended for weight loss
- Amount fed should target a 1% to 2% loss in body weight per week; faster weight loss may stimulate weight rebound once weight loss is achieved
- High moisture diets can be used to reduce calories per serving; this approach appears to be more effective for cats versus dogs, as cats tend to control their intake based on volume
- If the client is not willing to use a therapeutic diet, severe calorie restriction should be avoided; a food diary can be used to record current intake over several days—subsequently, the pet should be fed 10% to 20% less than it previously received
- Treats are often part of the owner-pet bond; complete avoidance of treats is a hurdle to compliance with weight loss programs—instead, offer a “treat allowance” of 10% of the daily calories and use low-calorie treats suitable for dogs or cats, as directed by your pet’s veterinarian



FOLLOW-UP CARE

PATIENT MONITORING

- Frequent communication is important during the weight management program
- Telephone call from clinic to the owner to address any minor questions and to reinforce the importance of the program
- Patient should be weighed in the clinic on a monthly basis; if needed, adjustments in food allowance guidelines should be made at this time
- Once the patient has achieved an ideal body condition score (BCS; estimate of weight status [under or overweight] as compared to normal weight) guidelines should be provided for weight maintenance; continue to measure food, monitor BCS or body weight, and adjust food allowance as needed to maintain the goal weight

PREVENTIONS AND AVOIDANCE

- Monitor food intake, weight, and body condition score (BCS; estimate of weight status [under or overweight] as compared to normal weight) throughout life to prevent weight gain and obesity
- Maintain a healthy diet and reduce caloric intake if pet starts gaining weight (even small weight gains of one-to-two pounds can be significant in small- and medium-size dogs and in cats)

POSSIBLE COMPLICATIONS

- Obesity leads to increased risk for diseases (such as osteoarthritis, diabetes mellitus) or shortened life span



KEY POINTS

- Obesity leads to increased risk for diseases (such as osteoarthritis, diabetes mellitus) or shortened life span
- Weight loss and maintenance of reduced weight depend on changes in the way the owner feeds and interacts with the pet
- Monitor food intake, weight, and body condition score (BCS; estimate of weight status [under or overweight] as compared to normal weight) throughout life to prevent weight gain and obesity
- Maintain a healthy diet and reduce caloric intake if pet starts gaining weight (even small weight gains of one-to-two pounds can be significant in small- and medium-size dogs and in cats)

GASTROINTESTINAL OBSTRUCTION

(BLOCKAGE OF THE GASTROINTESTINAL TRACT)



BASICS

OVERVIEW

- “Gastro-“ refers to stomach; “intestinal” refers to the intestines
- “Gastrointestinal obstruction” is a partial or complete blockage or obstruction of the flow of solid or liquid nutrients taken into the body (known as “ingesta”) and/or secretions from the stomach into the intestines and through the intestines

GENETICS

- Unknown

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Foreign bodies more common in dogs

Breed Predispositions

- Congenital (present at birth) narrowing of the area where the stomach and upper small intestine join together (area is the “pylorus;” condition known as “pyloric stenosis”)—more common in short-nosed, flat-faced (known as “brachycephalic”) breeds (such as boxers, Boston terriers) and Siamese cats
- Acquired (condition that develops sometime later in life/after birth) long-term (chronic) disease characterized by thickening of the stomach at the area where the stomach and upper small intestine join together (condition known as “hypertrophic pyloric gastropathy”)—more common in Lhasa apsos, shih tzus, Pekingese, and poodles
- Stomach dilates with gas and/or fluid (known as “gastric dilatation”), and subsequently rotates around its short axis (known as “volvulus”)—condition known as “gastric dilatation-volvulus” or “bloat”—more common in large-breed dogs (such as German shepherd dogs, Great Danes)

Mean Age and Range

- Foreign bodies—more common in young animals, but can occur at any age
- Pyloric stenosis (narrowing of the area where the stomach and upper small intestine join together)—occurs most often in young animals
- Long-term (chronic) hypertrophic pyloric gastropathy (disease characterized by thickening of the stomach at the area where the stomach and upper small intestine join together)—more common in middle-aged and older animals
- Folding of one segment of the intestine into another segment (known as “intussusception”)—most common in young animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vomiting—hallmark sign; may occur soon after eating, especially with blockage at the area where the stomach empties into the upper small intestine (known as “gastric outlet obstruction”); may be characterized as “projectile vomiting”
- Other variable clinical signs—lack of appetite (known as “anorexia”); sluggishness (lethargy); general signs of discomfort and “not feeling well” (known as “malaise”); excessive salivation (known as “ptyalism”); diarrhea; black, tarry stools (due to the presence of digested blood; condition known as “melena”); and weight loss
- Even if the animal is continuing to have bowel movements, this does not rule-out intestinal obstruction
- Physical examination findings can vary from “normal” to animal in “life-threatening crisis”—signs may include dehydration, shock, presence of a foreign body, abdominal discomfort or pain, and abdominal mass
- Linear foreign bodies (such as string)—careful examination under the tongue is essential for detection; although more common in cats, linear foreign bodies occur in dogs

CAUSES

Gastric Outflow Obstruction (blockage at the area where the stomach empties into the upper small intestine)

- Foreign bodies
- Pyloric stenosis (narrowing of the area where the stomach and upper small intestine join together)
- Long-term (chronic) hypertrophic pyloric gastropathy (disease characterized by thickening of the stomach at the area where the stomach and upper small intestine join together)
- Tumor or cancer

- Stomach dilates with gas and/or fluid (gastric dilatation), and subsequently rotates around its short axis (volvulus)—condition known as “gastric dilatation-volvulus” or “bloat”
- Inflammation of the stomach and/or intestines, characterized by the presence of nodules (known as “granulomatous gastritis” [stomach] or “granulomatous gastroenteritis” [stomach and intestines]), such as pythiosis (infection caused by *Pythium*, a water mold)

Small Intestinal Obstruction

- Foreign bodies
- Folding of one segment of the intestine into another segment (intussusception)
- Defect or tear in the muscular wall of the abdomen, allowing intestines to slide into an abnormal location and become trapped (known as a “hernia with incarceration”)
- Twisting of the support tissues of the intestines (known as “mesenteric torsion or volvulus”)
- Tumor or cancer
- Inflammation of the intestines, characterized by the presence of nodules (known as “granulomatous enteritis”)
- Abnormal narrowing of the small intestine (known as an “intestinal stricture”)

RISK FACTORS

- Exposure to and tendency to eat foreign bodies (such as rocks, string, or cloth)
- Folding of one segment of the intestine into another segment (intussusception)—associated with intestinal parasites and viral infection of the intestines (known as “viral enteritis,” such as parvovirus infection)



TREATMENT

HEALTH CARE

- Inpatient—for diagnosis, initial supportive medical care, and relief of the blockage or obstruction (usually with surgery)
- Delay in diagnosis may result in death of intestinal tissue (known as “intestinal necrosis”), abnormal opening or hole in the stomach or intestines (known as a “perforation”), and bacterial infection of the lining of the abdomen (known as “septic peritonitis”)
- Intravenous fluids—necessary to treat dehydration, to provide circulatory support, and to correct acid–base and electrolyte abnormalities
- Colloids may be beneficial; “colloids” are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch
- Potassium supplementation

ACTIVITY

- Restricted

DIET

- Nothing by mouth until relief of blockage or obstruction and resolution of vomiting; then feed bland diet for 1 to 2 days, with gradual return to normal diet

SURGERY

- Surgery—sudden (acute) intestinal blockages or obstructions are emergencies, and surgery should be performed as soon as possible after immediate supportive medical care

Gastric Outflow Obstruction

- Surgical widening of the pylorus, the area where the stomach and upper small intestines join together (procedure known as a “pyloroplasty”) or a surgical incision into the muscle of the pylorus (procedure known as a “pyloromyotomy”)—for pyloric stenosis (narrowing of the area where the stomach and upper small intestine join together) or chronic hypertrophic pyloric gastropathy (disease characterized by thickening of the stomach at the area where the stomach and upper small intestine join together)
- Surgical incision into the stomach (known as a “gastrotomy”)—for foreign bodies that cannot be removed using a special lighted instrument called an “endoscope” that is passed into the esophagus and stomach through the mouth (general term for procedure is “endoscopy”)
- Surgical removal of a section of the stomach and upper small intestine—for nodular inflammation (granulomatous gastroenteritis) or masses
- Surgical attachment of the stomach to the abdominal wall (known as a “gastropexy”)—for gastric dilatation-volvulus or bloat (condition in which the stomach dilates with gas and/or fluid [gastric dilatation], and subsequently rotates around its short axis [volvulus])

Intestinal Obstruction

- Surgical incision into the intestines (known as an “enterotomy”)—used to remove intestinal foreign bodies
- Surgical removal of a section of the intestines, with connection of the ends of the remaining sections of the intestines (known as an “intestinal resection and anastomosis”)—used for treatment of reduced blood flow to part of the intestinal tract, usually due to some type of blockage in a blood vessel, leading to decreased oxygen in the tissues (condition known as “intestinal ischemia”) and subsequent death of intestinal tissues (intestinal necrosis)
- Opening into the abdomen to allow flushing of the abdomen and lining of the abdomen (known as “open peritoneal lavage”)—treatment of abnormal opening or hole in the stomach or intestines (perforation) and bacterial infection of the lining of the abdomen (septic peritonitis)
- Surgical attachment of the intestines to the abdominal wall (known as an “enteropexy”)—treatment of folding of one segment of the intestine into another segment (intussusception)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Broad-spectrum antibiotics—examples are ampicillin or ticarcillin/clavulanate and an aminoglycoside (such as gentamicin) or a fluoroquinolone (such as enrofloxacin)
- Short-acting steroids—for shock; such as dexamethasone sodium phosphate or prednisolone sodium succinate
- Medications to control nausea and vomiting (known as “antiemetics”)—metoclopramide; may be given *after* the blockage or obstruction has been relieved
- H₂-blockers (such as ranitidine) and/or stomach-lining protectants (such as sucralfate)—may be used in patients with ulcers of the stomach and/or intestines



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor hydration, packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood) and total solids (a quick laboratory test that provides general information on the level of protein in the fluid portion of the blood), and electrolyte (such as sodium, potassium, chloride) status closely; adjust fluid therapy accordingly
- Monitor postoperatively for signs of inflammation of the lining of the abdomen (peritonitis)

PREVENTIONS AND AVOIDANCE

- Some pets with tendencies to eat foreign bodies may do so repeatedly; therefore, keep the pet away from possible foreign bodies, if possible
- Efforts to prevent eating of foreign bodies are important

POSSIBLE COMPLICATIONS

- Aspiration pneumonia
- Bacterial infection of the lining of the abdomen (septic peritonitis)
- Death of intestinal tissue (intestinal necrosis)
- Abnormal opening or hole in the stomach or intestines (perforation)
- Splitting open or bursting along the incision line (known as “dehiscence”)
- Lack of normal intestinal motility (known as “ileus”) and/or weakened or decreased muscular movement of the stomach (known as “gastroparesis”)

EXPECTED COURSE AND PROGNOSIS

- Uncomplicated cases—prognosis good to excellent
- Abnormal opening or hole in the intestines (intestinal perforation) and bacterial infection of the lining of the abdomen (septic peritonitis)—prognosis guarded initially
- Blockage from inflammation of the stomach and/or intestines, characterized by the presence of nodules (obstructive granulomatous gastroenteritis)—prognosis guarded to poor, especially with pythiosis (infection caused by *Pythium*, a water mold)
- Twisting of the support tissues of the intestines (known as “mesenteric torsion or volvulus”)—prognosis poor to grave (most patients

die despite surgery)



KEY POINTS

- Animals with the tendency to eat foreign bodies often are repeat offenders; all reasonable efforts to prevent access to foreign bodies should be made
- Sudden (acute) intestinal blockages or obstructions are emergencies, and surgery should be performed as soon as possible after immediate supportive medical care

URINARY TRACT OBSTRUCTION



BASICS

OVERVIEW

- Restricted flow of urine at any point in the urinary tract, from the kidneys to the external urethral orifice (the opening through which urine passes during urination)
- The urinary tract consists of the kidneys, the ureters (the tubes running from the kidneys to the bladder), the urinary bladder (that collects urine and stores it until the animal urinates), and the urethra (the tube from the bladder to the outside, through which urine flows out of the body)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Predominant Sex

- More common in males than in females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Abnormal frequent passage of urine (known as “pollakiuria”)—common
- Straining with slow, painful discharge of urine (known as “stranguria”)
- Reduced velocity or caliber of the urine stream or no urine flow during efforts to empty the bladder (voiding efforts)
- Obvious blood in the urine (known as “gross hematuria”)
- Signs of excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) that develop when urinary tract obstruction is complete (or nearly complete): sluggishness (lethargy), dull attitude, reduced appetite, and vomiting
- Excessive urine in the bladder (causing an overly large or tense/turgid bladder) or inappropriate retained urine (urine remains after voiding efforts); distension of the urinary bladder can be felt during physical examination
- Urinary tract stones (known as “uroliths”) often can be felt in the urethra (the tube from the bladder to the outside, through which urine flows out of the body) of an obstructed male dog, during physical examination
- Occasionally, an enlarged kidney or kidneys may be felt during physical examination in an animal with long-term (chronic) partial blockage or obstruction of the ureter(s), especially when the lesion involves only one side; the ureter is the tube running from the kidney to the bladder
- Signs of severely excessive levels of urea and other nitrogenous waste products in the blood (uremia or azotemia)—dehydration; weakness; low body temperature (known as “hypothermia”); low heart rate (known as “bradycardia”) with moderately increased levels of potassium in the blood (known as “hyperkalemia”); high rate of shallow respirations; stupor or coma; seizures occurring terminally; rapid heart rate (known as “tachycardia”) resulting from irregular heartbeats induced by severely increased levels of potassium in the blood (hyperkalemia)
- Signs of rupture or perforation of the urinary tract—leakage of urine into the abdomen causes abdominal pain and distension; leakage of urine into tissues around the urethra (the tube from the bladder to the outside, through which urine flows out of the body) causes pain and swelling in the pelvis or in the tissue between the anus and vulva in the female or anus and scrotum in the male, depending on the site of the urethral injury; fever

CAUSES

Intraluminal Causes (blockage or obstruction involving the inner, open space of the tubular ureters and urethra)

- Solid or semisolid structures including urinary tract stones (uroliths); accumulations of minerals and inflammatory materials in a matrix (known as “urethral plugs”) in cats; blood clots; and sloughed tissue fragments
- Most common site—the urethra
- Urinary tract stones (urolithiasis)—most common cause in male dogs
- Accumulations of minerals and inflammatory materials in a matrix (urethral plugs)—most common cause in male cats

Intramural Causes (blockage or obstruction involving the wall of a hollow organ, such as the bladder)

- Tumors or cancer of the bladder neck (the junction between the bladder and the urethra) or urethra (the tube from the bladder to the outside, through which urine flows out of the body)—common cause in dogs
- Nodular inflammatory lesions, characterized by the presence of pus (known as “pyogranulomatous inflammatory lesions”) in the urethra—seen occasionally in dogs
- Scar tissue (known as “fibrosis”) at a site of prior injury or inflammation can cause narrowing (stricture or stenosis), which may impede urine flow or may be a site where debris becomes lodged within the lumen (the inner open space of the ureter [the tube from the kidney

to the bladder] or urethra [the tube from the bladder to the outside, through which urine flows out of the body])

- Prostatic disorders in male dogs
- Fluid build-up (known as “edema”), bleeding, or spasm of muscular components can occur at sites of blockage or obstruction involving the inner, open space of the tubular ureters or urethra (intraluminal obstruction), and contribute to persistent or recurrent obstruction to urinary flow after removal of the intraluminal blockage; tissue changes might develop because of injury inflicted by the obstructing material, by the manipulations used to remove the obstructing material, or both
- Ruptures, lacerations, and punctures—usually caused by traumatic incidents

Miscellaneous Causes

- Displacement of the urinary bladder into a perineal hernia; a perineal hernia develops when the muscles supporting the rectum weaken and separate, allowing the rectum and/or bladder to slide under the skin and causing swelling in the area of the anus
- Nervous system disorders

RISK FACTORS

- Urinary tract stones (urolithiasis), particularly in males
- Feline lower urinary tract disease (FLUTD), particularly in males
- Prostatic disease in male dogs



TREATMENT

HEALTH CARE

- Complete urinary tract obstruction is a medical emergency that can be life threatening; treatment usually should be started immediately
- Partial urinary tract obstruction—not necessarily an emergency, but these patients may be at risk for developing complete obstruction; may cause irreversible urinary tract damage, if not treated promptly
- Treat as an inpatient until the patient’s ability to urinate has been restored
- Treatment has three major components: 1) combating the problems associated with excess levels of urea and other nitrogenous waste products in the blood that build up due to the urinary tract obstruction (known as “postrenal uremia”)—problems include dehydration; low body temperature (hypothermia); accumulation of acidic compounds in the body (known as “acidosis”); increased levels of potassium in the blood (hyperkalemia); 2) restoring and maintaining an open pathway for urine outflow; and 3) implementing specific treatment for the underlying cause of urine retention and urinary tract obstruction
- Give fluid therapy to patients with dehydration or with excessive levels of urea and other nitrogenous waste products in the blood (uremia or azotemia)
- When substantial generalized (systemic) problems exist, start fluid administration and other supportive measures first; careful decompression by tapping the bladder to remove urine (known as “cystocentesis”) may be performed before anesthesia and catheterization

SURGERY

- Surgery is required sometimes
- Urinary diversion by tube cystostomy is useful in selected cases; tube cystostomy is surgical placement of a catheter from the bladder and exiting through the abdominal wall to allow urine to be removed from the body, thus bypassing blockage of the urethra



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Procedures for relief of obstruction often require, or are facilitated by, giving sedatives or anesthetics; generally isoflurane is the anesthetic of choice; however, a variety of other anesthetics or sedatives can give satisfactory results



FOLLOW-UP CARE

PATIENT MONITORING

- Assess urine production and hydration status frequently, and adjust fluid administration rate accordingly
- Verify ability to urinate adequately or use urinary catheterization to combat urine retention
- If catheter insertion requires repeated use of sedatives or anesthetics or is unduly traumatic, indwelling catheterization with closed drainage is appropriate; however, frequent brief catheterization may be a better choice, if the catheter can be inserted easily on a repeated basis
- When the electrocardiogram (ECG) indicates changes in the heart rhythm that potentially are life-threatening, use continuous monitoring to guide treatment and evaluate response

POSSIBLE COMPLICATIONS

- Death
- Injury to the urinary tract, while trying to relieve obstruction
- Low levels of potassium in the blood (hypokalemia) during postobstruction diuresis (a condition in which the body produces large volumes of urine after a urinary tract blockage has been relieved)
- Recurrence of obstruction

EXPECTED COURSE AND PROGNOSIS

- Long-term management and prognosis depend on the cause of the blockage or obstruction



KEY POINTS

- Urinary tract obstruction is restricted flow of urine at any point in the urinary tract, from the kidneys to the external urethral orifice, the opening through which urine passes during urination
- Complete urinary tract obstruction is a medical emergency that can be life threatening; treatment usually should be started immediately
- Partial urinary tract obstruction—not necessarily an emergency, but these patients may be at risk for developing complete obstruction; may cause irreversible urinary tract damage, if not treated promptly
- Long-term management and prognosis depend on the cause of the blockage or obstruction

ODONTOCLASTIC RESORPTIVE LESIONS—CATS



BASICS

OVERVIEW

- Loss of varying amounts of substance of the tooth by a disease process (known as “dental resorptions”) affecting cats
- “Odontoclastic” refers to “odontoclasts,” which are cells found around the teeth and are believed to lead to resorption (loss of substance) of the teeth
- A relatively newly recognized syndrome
- Also known as “FORL” for feline odontoclastic resorptive lesion

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cat

Breed Predispositions

- Asian short-haired cats, Siamese, Persian, and Abyssinian may show breed susceptibilities

Mean Age and Range

- Nearly 50% of cats older than five years old will have at least one FORL
- Likelihood of FORL increases as the cat ages

SIGNS/OBSERVED CHANGES in the ANIMAL

- Most affected cats do not show clinical signs; some show excessive salivation/drooling (known as “hypersalivation”); bleeding from the mouth or difficulty chewing; some cats pick up and drop food (especially hard food) when eating; others hiss while chewing.
- Some cats have behavior changes—they may hide or become aggressive
- Pain, evidenced by jaw spasms
- Tartar or calculus (mineralized plaque on the tooth surface) and excessive gum tissue (known as “hyperplastic gingival tissue”) may cover or hide the FORL
- FORLs can be found on any tooth; most commonly affected are the mandibular (lower jaw) third premolar and molar teeth, followed by the maxillary (upper jaw) third and fourth premolar teeth
- FORLs are classified Stage 1-5, based on their depth and amount of tooth destruction as follows:
 - Stage 1 FORL—defect in the tooth is less than 0.5 mm deep
 - Stage 2 FORL—penetrates the dentin (hard portion of the tooth, surrounding the pulp [blood vessels and nerves] and covered by enamel), but does not enter the endodontic system (internal part of the tooth containing the blood vessels and nerves; also known as the “pulp”); the extent of root involvement (determined on dental X-rays) helps to determine therapy
 - Stage 3 FORL—penetrates into the endodontic system (internal part of the tooth containing the blood vessels and nerves)
 - Stage 4 FORL—substantial structural damage to roots (part of the tooth below the gum line) and crown (part of the tooth above the gum line)
 - Stage 5 FORL—the crown (part of the tooth above the gum line) is gone; swelling of the gum tissue covers the retained root

CAUSES

- Unknown; likely many factors contribute to development of FORLs
- Affected cats may have calcium-regulation problems; an improper ratio of dietary calcium, magnesium, and phosphorus; or parathyroid-gland malfunction, producing calcium imbalance
- Hyperreactivity to inflammatory cells, dental plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white blood cells, food particles, and components of saliva), and/or tartar or calculus (mineralized plaque on the tooth surface); various compounds (endotoxins; prostaglandins, cytokines, and proteinases) also are under investigation as possible causes



TREATMENT

DIET

- Add water to diet to soften food

SURGERY

- Stage 1 FORLs—an enamel defect is noted; the lesion is minimally sensitive because it has not penetrated the dentin (hard portion of the tooth, surrounding the pulp [blood vessels and nerves] and covered by enamel); therapy includes thorough cleaning and polishing and possible surgical removal of some gum tissue (known as “gingivectomy”) and surgical contouring of the tooth surface (known as “odontoplasty”)
- Stage 2 FORLs—penetrate the dentin (hard portion of the tooth, surrounding the pulp [blood vessels and nerves] and covered by enamel); often require either extraction or crown (part of the tooth above the gum line) reduction
- Stage 3 FORLs—enter the endodontic system (internal part of the tooth containing the blood vessels and nerves; also known as the “pulp”); require either extraction or crown (part of the tooth above the gum line) reduction
- Stage 4 FORLs—the crown (part of the tooth above the gum line) is eroded or fractured with part of the crown remaining; gum tissue (gingiva) grows over the root fragments, yielding a sensitive bleeding lesion upon probing; additional extraction may be needed
- Stage 5 FORLs—the crown (part of the tooth above the gum line) is gone and roots remain; surgically remove any inflamed areas of tissue



KEY POINTS

- Loss of varying amounts of substance of the tooth by a disease process (known as “dental resorptions”) affecting cats
- Nearly 50% of cats older than five years old will have at least one FORL
- Likelihood of FORL increases as the cat ages
- Daily home brushing may help control plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white blood cells, food particles, and components of saliva)

TUMORS OR MASSES IN THE MOUTH (ORAL MASSES)



BASICS

OVERVIEW

- “Oral” refers to the mouth; “oral masses” are tumors or growths located in the mouth
- Oral masses may be benign or malignant (that is, cancer)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Golden retrievers, German shorthaired pointers, Weimaraners, St. Bernards, and cocker spaniels are more prone to tumors of the mouth than other breeds; dachshunds and beagles are less prone to tumors of the mouth than other breeds; boxers are more prone to have enlargement of the gums (known as “gingival hyperplasia”) than other breeds
- Malignant melanoma—the most common cancer of the mouth in the dog; cocker spaniels, German shepherd dogs, chow chows, and dogs with heavily pigmented linings of the mouth (known as “mucous membranes”) are more likely to develop malignant melanoma than other dogs
- Squamous cell carcinoma—the second most common cancer of the mouth in the dog; large-breed dogs are more likely to develop squamous cell carcinoma than other dogs

Mean Age and Range

- Older animals are affected most often
- Fibromatous epulis—the epulides (plural of epulis) are masses located on the gums; they are the most common benign tumor of the mouth; fibromatous epulis age range is 1 to 17 years; mean age is 7.5 years
- Papillary squamous cell carcinoma—a rapidly growing cancer of young dogs (less than 1 year of age)
- Fibrosarcomas—the third most common cancer of the mouth in dogs; seen in large, older male dogs
- Squamous cell carcinoma in the cat—type of cancer in the mouth; age range is 3 to 21 years; mean age is 12.5 years
- Fibrosarcoma in the cat—type of cancer in the mouth; age range is 1 to 21 years; mean age is 10.3 years

Predominant Sex

- Malignant melanoma—the most common cancer of the mouth in the dog; males more frequently affected than females
- Fibrosarcomas—the third most common cancer of the mouth in dogs; seen in large, older male dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- May have no signs
- May include bad breath (known as “halitosis”), tooth displacement, malocclusion (any deviation in the relationship or contact between the biting and chewing surfaces of the upper and lower teeth), bleeding in the mouth, drooling, and reluctance to chew

RISK FACTORS

- Squamous cell carcinoma of the tonsils occurs ten times more commonly in dogs from urban settings than in rural dogs
- Squamous cell carcinoma—more common in white dogs in one study
- Any long-term (chronic) source of irritation to the tissues of the mouth (such as inflammation/infection of the tissues surrounding and supporting the teeth [known as “periodontal disease”] or second-hand smoke) increases the risk of tumor development in the mouth
- Feline leukemia virus (FeLV) or feline immunodeficiency virus (FIV) may play a role in squamous cell carcinoma development in cats
- Some researchers showed that cats that wore flea collars had 5 times the risk of developing squamous cell carcinoma of the mouth than cats that did not wear flea collars



TREATMENT

HEALTH CARE

- Depends on the tumor type

- Benign tumors are treatable with long-term success via surgery
- Cancer (malignant tumors) is treated surgically with varying success, depending on tumor type, location, and if the cancer has spread to other tissues of the body (known as “metastasis”) at time of presentation to the pet’s veterinarian
- In advanced cancer, combined therapy (surgery, chemotherapy, and radiation) may provide the best care

DIET

- Gruel or liquefied diet may be necessary following surgery of the mouth
- Tube feeding may be necessary

SURGERY

- Fibromatous epulis—surgical removal is the treatment of choice; freezing (known as “cryotherapy”); and radiation treatment also give long-term success
- Peripheral odontogenic fibromas (ossifying epulis)—treat the same as fibromatous epulis
- Acanthomatous ameloblastoma—surgical removal is usually curative; radiation also has been used successfully; the combination of surgery and radiation may be most effective (requiring less aggressive surgery), but if radiation is not readily available, surgery may be the only option; multiple injections of bleomycin at the tumor site have been effective in a small number of reported cases
- Malignant melanoma—if surgery is chosen for therapy, it should be aggressive; typically involving surgical removal of the lower jaw or mandible (known as “mandibulectomy”) or the upper jaw or maxilla (known as “maxillectomy”)
- Squamous cell carcinoma—may be removed surgically with wide margins or may be treated with radiation therapy in the dog, especially; surgical removal of the lower jaw or mandible (mandibulectomy) or the upper jaw or maxilla (maxillectomy) with a 2-cm clean surgical margin is the goal; dogs tolerate surgical removal of 40% to 60% of the tongue (known as “partial glossotomy”); surgery, radiation, and chemotherapy (mitoxantrone) may be the best options for tumors larger than 2 cm or those with incomplete surgical removal
- Fibrosarcoma—usually requires surgical removal of the lower jaw or mandible (mandibulectomy) or the upper jaw or maxilla (maxillectomy)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Chemotherapy may be indicated for some forms of cancer in the mouth; chemotherapeutic drugs may include mitoxantrone, bleomycin, or cisplatin



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on type of tumor and the presence or absence of spread of cancer (metastasis)

PREVENTIONS AND AVOIDANCE

- Remove or treat any source of irritation to the tissues of the mouth (such as inflammation/infection of the tissues surrounding and supporting the teeth [periodontal disease] or second-hand smoke)

POSSIBLE COMPLICATIONS

- Surgical removal of part of the tongue may result in loss of blood supply to the remaining tongue, with death of tongue tissue
- Postoperative complications of surgical removal of the lower jaw or mandible (mandibulectomy) include splitting open or bursting along the incision line (known as “wound dehiscence”), difficulty grasping food, tongue hanging out of the mouth, and excessive drooling
- Surgical removal of the lower jaw or mandible (mandibulectomy) can be performed in cats, but mandibulectomy results in greater complications (such as tongue swelling) than in dogs
- With low-dose radiation therapy, diarrhea, nausea, vomiting, and hair loss may occur (regrowth of hair is usually white); high-dose radiation therapy has these complications as well as superficial loss of tissue on the surface of the lining of the mouth, frequently with inflammation (known as “oral ulceration”) and/or death of tissues in the mouth (known as “oral necrosis”), cataracts (opacities in the normally clear lens), and radiation-induced tumors (mainly in young dogs that underwent radiation therapy)
- Chemotherapy complications vary depending on the drug used

EXPECTED COURSE AND PROGNOSIS

- Dogs with inadequate tumor-free surgical margins were 2.5 times more likely to die of the tumor than those with complete surgical removal of the tumor (as demonstrated by microscopic evaluation of tumor margins); some surgical patients need feeding tubes to facilitate nutritional supplementation during the treatment period
- Dogs with tumors located behind the first premolar tooth had three times greater risk of dying from the disease than those with tumors located in front of the first premolar tooth
- Malignant melanoma—prognosis improves if the tumor is small and located in the front part of the lower jaw or mandible; treatment of malignant melanoma involves surgical removal of the lower jaw or mandible (mandibulectomy) or the upper jaw or maxilla (maxillectomy)—median survival times average 8 months; combination of surgery, radiation, and chemotherapy (low-dose cisplatin) yielded a median survival of 14 months in one study; pigmentation does not affect the prognosis; this cancer of the mouth is relatively resistant to radiation therapy—one study showed a median survival time of 14 months after radiation only; the problem with melanoma is not local disease management, but spread of the cancer to other body tissues (metastasis)
- Vaccination of dogs with malignant melanoma seems to be curative and is offered at a number of sites throughout North America
- Squamous cell carcinoma—better long-term prognosis than malignant melanoma or fibrosarcoma in the dog; the prognosis is better if the cancer is located toward the front of the mouth than if it is located toward the back of the mouth in dogs; radiation therapy alone delivers a median survival rate of 15 to 17 months; in dogs, prognosis for survival following treatment for squamous cell carcinoma of the tongue is poor
- Fibrosarcoma—surgical removal of the cancer, with at least 2-cm margins, usually results in a 12-month median survival rate; surgical excision in combination with radiation therapy and chemotherapy offers the best prognosis; radiation or chemotherapy alone offered a poorer median survival rate than surgery alone; fibrosarcomas involving the roof of the mouth (palate) carry the poorest prognosis because of the inability to remove them adequately with surgery



KEY POINTS

- Oral masses are tumors or growths located in the mouth
- Oral masses may be benign or malignant (that is, cancer)
- Benign tumors are treatable with long-term success via surgery
- Cancer (malignant tumors) is treated surgically with varying success, depending on tumor type, location, and if the cancer has spread to other tissues of the body (metastasis)

ORAL ULCERATION AND CHRONIC ULCERATIVE PARADENTAL STOMATITIS

(ULCERS OF THE MOUTH AND INFLAMMATION AROUND THE TEETH)



BASICS

OVERVIEW

- “Oral ulceration” is the term for “ulcers of the mouth;” ulcers are lesions on the moist tissues, characterized by the loss of the top layer(s) of tissue, usually associated with inflammation
- “Paradental” refers to adjacent, beside, or alongside (“para-”) the teeth (“dental”)
- “Stomatitis” is inflammation of the lining of the mouth
- “Chronic” is defined as being long-term or prolonged
- Oral ulceration and chronic ulcerative paradental stomatitis are conditions involving localized (“focal”) loss or multiple areas (“multifocal”) of loss of the top layer(s) of the lining of the mouth
- Chronic ulcerative paradental stomatitis is also known as “CUPS”
- “Lymphocytic plasmacytic stomatitis” is seen in cats; it also is known as “LPS”—it is inflammation of the lining of the mouth, characterized by the presence of lymphocytes and plasma cells; lymphocytes are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process; plasma cells or plasmacytes are a specialized type of white-blood cell; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Inflammation of the lining of the mouth, characterized by ulcers (known as “ulcerative stomatitis” and also known as “chronic ulcerative paradental stomatitis” [CUPS])—Maltese, Cavalier King Charles spaniels, cocker spaniels, Bouvier des Flandres
- Inflammation/infection of the bone for unknown reason (known as “idiopathic osteomyelitis”)—cocker spaniels may have increased likelihood of developing idiopathic osteomyelitis as compared to other dog breeds; complication associated with CUPS
- Inflammation of the lining of the mouth, characterized by the presence of lymphocytes and plasma cells in cats (known as “feline stomatitis” or “lymphocytic plasmacytic stomatitis” [LPS])—the Somali and Abyssinian may have a tendency to develop feline stomatitis

Mean Age and Range

- Any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Bad breath (known as “halitosis”)
- Inflammation of the gums (known as “gingivitis”)
- Inflammation of the area between the cavity or back of the mouth and the throat (area known as the “fauces; inflammation known as “faucitis”)
- Inflammation of the throat or pharynx (known as “pharyngitis”)
- Inflammation of the lining of the cheek (known as “buccitis”) with ulcers (known as “buccal mucosal ulceration”)
- Excessive salivation (known as “hypersalivation”) with thick, ropery saliva
- Pain
- Lack of appetite (known as “anorexia”)
- Ulcers of the lining of the mouth (known as “mucosal ulceration”)—ulcers that occur on surfaces of the moist lining of the mouth that oppose the teeth (known as “kissing ulcers”) common in CUPS
- Plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva)—with or without tartar or calculus (mineralized plaque on the tooth surface)
- Exposed, dead (necrotic) bone—with inflammation of the bone of the tooth socket (known as “alveolar osteitis”) and inflammation/infection of the bone for unknown reason (idiopathic osteomyelitis)
- Behavior changes secondary to pain or sensitivity in the mouth

- Scar formation on lateral margins of tongue—with CUPS

CAUSES

Metabolic

- Diabetes mellitus (“sugar diabetes”)
- Inadequate production of parathyroid hormone by the parathyroid glands (known as “hypoparathyroidism”)
- Inadequate production of thyroid hormone (known as “hypothyroidism”)
- Kidney disease/failure—excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”)

Nutritional

- Protein-calorie malnutrition
- Riboflavin deficiency; riboflavin is part of the vitamin B complex

Cancer

- Dog—malignant melanoma; squamous cell carcinoma; fibrosarcoma
- Cat—squamous cell carcinoma; fibrosarcoma; malignant melanoma

Immune-mediated

- Autoimmune diseases (such as pemphigus vulgaris, bullous pemphigoid, systemic lupus erythematosus, discoid lupus erythematosus)
- Drug-induced—ulcerative disorder of the skin and moist tissues of the mouth (known as “toxic epidermal necrolysis”)
- Immune-mediated inflammation of the blood vessels (known as “immune-mediated vasculitis”)

Infectious

- Retrovirus—cats; feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV)
- Calicivirus—cat
- Herpesvirus—cat
- Leptospirosis—dog
- Inflammation/infection of the tissues surrounding and supporting the teeth (known as “periodontal disease”)—dog and cat

Traumatic

- Foreign body—bone or wood fragments
- Electric-cord shock
- Any deviation in the relationship or contact between the biting and chewing surfaces of the upper and lower teeth (known as “malocclusion”)
- “Gum-chewer’s disease”—chronic chewing of the moist tissues lining the cheek

Chemical/Toxic

- Acids
- Thallium

Idiopathic (Unknown Cause)

- Eosinophilic granuloma (a mass or nodular lesion containing a type of white-blood cell, called an eosinophil)—cats, Siberian huskies, Samoyeds
- Lymphocytic plasmacytic stomatitis (LPS)—cats
- Chronic ulcerative paradental stomatitis (CUPS)—dogs; allergic reaction to plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva)
- Inflammation/infection of the bone for unknown reason (idiopathic osteomyelitis)—dogs



TREATMENT

HEALTH CARE

- Supportive therapy—soft diet; fluids; hospitalization in severe cases
- Chronic ulcerative paradental stomatitis (CUPS)—continuous, meticulous home care to prevent plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva) accumulation; dental cleaning initially and frequently; periodontal therapy; extraction of diseased teeth
- Underlying metabolic or other disease—treat underlying illness

DIET

- Soft diet
- Nutritional support—via feeding tube

SURGERY

- Select extractions (partial mouth, teeth in the back of the mouth, or full mouth)—may be indicated for long-term (chronic) conditions of unknown cause (idiopathic), such as CUPS and LPS, to remove the source of reaction (plaque [the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva] and teeth)
- Removal of entire tooth structure—important in extraction treatment for LPS
- Removal of dead (necrotic) bone or bone that has lost blood supply (known as “avascular bone”) indicated for inflammation/infection of the bone for unknown reason (idiopathic osteomyelitis)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics—treat primary and secondary bacterial infections—clindamycin; amoxicillin-clavulanate; tetracycline
- Broad-spectrum antibiotics—indicated for inflammation/infection of the bone for unknown reason (idiopathic osteomyelitis)
- Anti-inflammatory/immunosuppressive drugs—used to decrease inflammation and to decrease the immune response; the comfort of the patient must be weighed against potential long-term side effects of steroid usage—prednisone
- Agents to protect the surface of the lining of the mouth (known as “mucosal protectants”) for chemical insults—sucralfate; cimetidine
- Pain relievers (known as “analgesics”) following extraction or teeth—carprofen; hydrocodone; tramadol
- Topical treatment (that is, treatment applied directly to the tissues of the mouth)—chlorhexidine solution or gel (antibacterial); zinc gluconate/ascorbic acid; stabilized chlorine dioxide for bad breath (halitosis)



FOLLOW-UP CARE

PATIENT MONITORING

- Inflammation may take 4 to 6 weeks to subside after extractions due to plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva) build-up on sutures and the tongue

EXPECTED COURSE AND PROGNOSIS

- Prognosis is guarded, response to therapy depends on underlying cause, and prolonged treatment and/or further extractions may be necessary



KEY POINTS

- Inflammation may take 4 to 6 weeks to subside after extractions
- Prognosis is guarded, response to therapy depends on underlying cause, and prolonged treatment and/or further extractions may be necessary
- Any level of home care (brushing or topical antimicrobials) that can be provided is encouraged in chronic ulcerative paradental stomatitis (CUPS) or lymphocytic plasmacytic stomatitis (LPS)

ORGANOPHOSPHATE AND CARBAMATE TOXICITY

(TYPES OF INSECTICIDE POISONING)



BASICS

OVERVIEW

- Organophosphates and carbamates are insecticides used to control insects on animals and plants, around the home and yard, and in agricultural settings
- Organophosphates and carbamates decrease the activity of acetylcholinesterase, an enzyme that breaks down acetylcholine; acetylcholine is a chemical that transmits information from the autonomic nervous system to various organs in the body (such as the heart, blood vessels, and gastrointestinal tract); decreased levels of the enzyme lead to excessive amounts of acetylcholine being present, resulting in “over stimulation” of the target organs
- The autonomic nervous system is involved in the control of muscles in the heart, blood vessels, gastrointestinal tract, and other organs; it is composed of two parts—the sympathetic and the parasympathetic parts; the two parts cause opposing responses; for example, the sympathetic nervous system speeds up the heart and causes the blood vessels to constrict or become small while the parasympathetic nervous system slows the heart and causes the blood vessels to expand or dilate
- Organophosphate and carbamate toxicity results from exposure to organophosphorous compounds or carbamates
- From 2003 to 2005, the ASPCA Animal Poison Control Center experienced a 46% decrease in calls regarding organophosphates—this decrease likely is related to the federal Environmental Protection Agency (EPA) cancellations of various registrations of some organophosphate insecticides and approval of new formulations; however, canceled products often remain for years in homes and businesses; carbamate inquiries increased 15% during the same period
- Products intended for use on or around animals—organophosphate: chlorpyrifos, coumaphos, cythioate, diazinon, famphur, fenthion, phosmet, and tetrachlorvinphos; carbamate: carbaryl and propoxur
- Products intended for use in agriculture and lawn and gardens—organophosphate: acephate, chlorpyrifos, diazinon, disulfoton, fonofos, malathion, parathion, terbufos, and others; carbamate: carbofuran and methomyl

GENETICS

- Animals with inherently low levels or activity of the enzyme that breaks down acetylcholine (that is, has low cholinesterase activity)—more susceptible to cholinesterase depression
- Cholinesterase activity—more easily inhibited in cats than in dogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats and small or exceptionally lean dogs—most susceptible

Breed Predilections

- Lean dogs (such as sight hounds and racing breeds) and lean longhair cats—more susceptible to cholinesterase inhibition because of lack of fat; many organophosphorous compounds and their breakdown products (known as “metabolites”) are stored in fat and slowly released into circulation

Mean Age and Range

- Young animals—more likely to become poisoned due to lower ability to diminish or remove (detoxify) the poison from their bodies

Predominant Sex

- Intact males more susceptible to some organophosphates

SIGNS/OBSERVED CHANGES in the ANIMAL

- Parasympathetic nervous system stimulation usually predominates, causing such signs as slowing of the heart and dilated blood vessels
- History often discloses heavy or repeated applications of flea and tick insecticides; evidence of exposure to an agricultural or home and garden product
- Carbamate insecticides (methomyl and carbofuran)—may cause rapid onset of seizures and breathing (respiratory) failure; treat aggressively without delay
- Organophosphate insecticides (cats, especially chlorpyrifos)—long-term (chronic) lack of appetite (anorexia), muscle weakness, and muscle twitching, with or without episodes of sudden (acute) poisoning (toxicosis), which may last for days to weeks
- Excessive salivation (known as “hypersalivation”)
- Vomiting
- Diarrhea

- Constricted or small pupils (known as “miosis”)
- Slow heart rate (known as “bradycardia”)
- Depression
- Wobbly gait (known as “ataxia”)
- Muscle tremors
- Seizures
- Increased body temperature (known as “hyperthermia”)
- Difficulty breathing (known as “dyspnea”)
- Breathing (respiratory) failure
- Death
- Patient may not exhibit all signs
- If sympathetic nervous system stimulation predominates—may result in lack of specific expected signs; signs may be reversed or opposite of expected (as described in preceding list)

CAUSES

- Overuse, misuse, or use of multiple organophosphate or carbamate insecticides
- Misuse of organophosphate insecticides in cats
- Intentional application of house or yard insecticides to the skin of animals

RISK FACTORS

- Concurrent exposure to multiple organophosphate- and/or carbamate-containing products
- Exposure to floors that are damp with organophosphorous premise products
- Incorrect dilution of insecticides
- Organophosphate-containing dips *labeled for dogs only*—inappropriately applied to cats



TREATMENT

HEALTH CARE

- Outpatient—mild signs from exposure to flea and tick collars and powders; treated by simply removing the collar or brushing excess powder from the coat
- Inpatient—continued salivation, tremors, or difficulty breathing (dyspnea)
- Basics of care—stabilization of patient; decontamination to remove source of exposure to insecticide; antidotal treatment with atropine (and pralidoxime chloride for organophosphate poisoning); supportive care
- Oxygen—if necessary, until breathing (respiration) returns to normal
- Fluid therapy—may be needed in cats that are not eating (anorectic)
- Bathing (for skin [dermal] exposure)—rinse with large volumes of water

DIET

- Cats that have long-term (chronic) lack of appetite (anorectic cats)—maintain nutritional and fluid requirements



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Pentobarbital to control seizures
- Diazepam or phenobarbital—controls seizures
- Atropine sulfate—antidote; administered immediately; repeated only as needed to control life-threatening clinical signs from parasympathetic stimulation
- Pralidoxime chloride or 2-PAM (Protopam®)—antidote; reduces muscle twitching; most beneficial against organophosphorous

insecticides when started within 24 hours of exposure; even several days after skin (dermal) exposure may stimulate cats that are not eating (anorexic cats) with or without muscle tremors to resume eating

- If animal has history of ingesting a liquid insecticidal solution—avoid inducing vomiting (emesis) due to the risk of aspiration, because many solutions contain hydrocarbon solvents; your pet’s veterinarian and/or Animal Poison Control should provide guidelines if inducing vomiting is appropriate
- If the animal has no clinical signs and the insecticide ingested was not liquid, induce vomiting with 3% hydrogen peroxide after feeding a moist meal; your pet’s veterinarian and/or Animal Poison Control should provide guidelines if inducing vomiting is appropriate
- Evacuation of the stomach for patient with clinical signs—flush the stomach (known as “gastric lavage”) with the patient intubated, under anesthesia, with a large-bore stomach tube; then administer activated charcoal containing sorbitol as a cathartic (to clean out the intestinal tract) in a water slurry
- Diarrhea—do not administer sorbitol-containing products



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor heart rate, breathing (respiration), and fluid and caloric intake

PREVENTIONS AND AVOIDANCE

- Closely follow directions on labels of insecticides—be especially careful to use products *only* on animals for which they are indicated (that is, use “dog only” products on dogs and *do not* use “dog only” products on cats)
- Avoid use on sick or debilitated animals
- Avoid simultaneous use of organophosphate and carbamate products

POSSIBLE COMPLICATIONS

- Death

EXPECTED COURSE AND PROGNOSIS

- Long-term (chronic) organophosphate insecticide–induced weakness and lack of appetite (anorexia) in cats—signs may last 2 to 4 weeks; most patients fully recover with aggressive nursing care
- Sudden poisoning (acute toxicosis) treated promptly—good prognosis



KEY POINTS

- Follow insecticide label directions carefully; read entire label before using insecticide
- Be especially careful to use products *only* on animals for which they are indicated (that is, use “dog only” products on dogs and *do not* use “dog only” products on cats)
- Avoid use on sick or debilitated animals
- Avoid simultaneous use of organophosphate and carbamate products
- Cats with long-term (chronic) lack of appetite (anorexia) and weakness may need days to weeks of supportive care for full recovery

OSTEOCHONDROSIS

(ABNORMAL BONE FORMATION IN GROWING DOGS)



BASICS

OVERVIEW

- Long bones (such as the humerus, radius and ulna in the foreleg and the femur and tibia in the rear leg) have three sections: the end of the bone, known as the “epiphysis;” the shaft or long portion of the bone, known as the “diaphysis;” and the area that connects the end and the shaft of the bone, known as the “metaphysis”
- The metaphysis is the area where bone growth occurs in puppies; the long bones in the body grow in length at specific areas known as “growth plates;” these areas usually continue to produce bone until the bones are fully developed, at which time, no further growth is needed; the growth plates then “close” and become part of the hard bone
- Bone is formed by the replacement of calcified cartilage at the growth plates; the bone-forming cells (known as “osteoblasts”) form bone on the cartilage structure; this process is known as “endochondral ossification”
- “Osteochondrosis” is a disorder of bone formation in the growth plates (areas where bone grows in length in the young animal) of the bone; it is a disease process in growing cartilage, primarily characterized by a disturbance of endochondral ossification that leads to excessive retention of cartilage

GENETICS

- Multiple genes are involved (known as “polygenetic transmission”)—expression determined by an interaction of genetic and environmental factors
- Heritability index—depends on breed

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Demonstrated clinically—horses; pigs; broiler chickens; turkeys; people

Breed Predilections

- Frequent and serious problem in many dog breeds
- Large- and giant-breed dogs—Great Danes, Labrador retrievers, Newfoundlands, rottweilers, Bernese mountain dogs, English setters, Old English sheepdogs

Mean Age and Range

- Onset of clinical signs—typically 4 to 8 months of age
- Diagnosis—generally 4 to 18 months of age
- Signs of secondary degenerative joint disease (progressive and permanent deterioration of joint cartilage)—any age

Predominant Sex

- Shoulder osteochondrosis—males are twice as likely to develop shoulder osteochondrosis than females
- Osteochondrosis of the elbow, stifle, or hock—none

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on the affected joint(s) and coexistent degenerative joint disease (progressive and permanent deterioration of joint cartilage)
- Lameness—most common sign; sudden or subtle onset; slight, moderate, or severe; one or more limbs may be involved; becomes worse after exercise; duration of several weeks to months; pet may support little weight on the affected limb
- Pain—usually elicited on feeling the limb by flexing, extending, or rotating the involved joint
- Generally a weight-bearing lameness
- Fluid build-up in the joint (known as “joint effusion”)—common with osteochondrosis of the elbow, stifle, and hock
- Decrease in muscle mass (known as “muscle atrophy”)—consistent finding with long-term (chronic) lameness

CAUSES

- Developmental disorder
- Nutritional disorder

RISK FACTORS

- Rapid growth and weight gain
- Diet containing three times the recommended calcium levels



TREATMENT

HEALTH CARE

- Ice packing (known as “cryotherapy”) of affected joint—immediately following surgery; 5 to 10 minutes three times a day for 3 to 5 days, or as directed by your pet’s veterinarian
- Range-of-motion exercises—initiated as soon as patient can tolerate joint movement

ACTIVITY

- Restricted
- Avoid hard, concussive activities (such as running on concrete)
- Following surgery for osteochondritis dissecans or “OCD” (abnormal development of bone and cartilage, leading to a flap of cartilage within the joint)—limit activity for 4 to 6 weeks; encourage early, active movement of the affected joint(s)

DIET

- Weight control—decreases stress placed on affected joint(s)

SURGERY

- Osteochondrosis is a nonsurgical condition, unless a related bone fragment moves into an area that causes clinical signs, then surgical removal of the bone fragment is indicated
- May progress to osteochondritis dissecans (abnormal development of bone and cartilage, leading to a flap of cartilage within the joint) as the patient grows
- Surgical procedure cutting into or entering a joint (known as an “arthrotomy”) or using a special lighted instrument called an “arthroscope” (general term for procedure is “arthroscopy”) to allow the surgeon to see inside the joint—indicated for most dogs with osteochondritis dissecans (abnormal development of bone and cartilage, leading to a flap of cartilage within the joint)
- Shoulder—indicated for all osteochondritis dissecans (abnormal development of bone and cartilage, leading to a flap of cartilage within the joint) lesions; exploratory procedure indicated for pain and lameness with X-ray evidence of osteochondrosis
- Elbow—indicated for all osteochondritis dissecans (abnormal development of bone and cartilage, leading to a flap of cartilage within the joint) lesions; indicated to assess for other bone conditions
- Stifle—controversial; patients develop degenerative joint disease (progressive and permanent deterioration of joint cartilage) even with surgical procedure; using a special lighted instrument called an “arthroscope” (general term for procedure is “arthroscopy”) to allow the surgeon to see inside the joint may improve the recovery rate and long-term function
- Hock—remove osteochondral flap; controversial; all patients develop severe degenerative joint disease (progressive and permanent deterioration of joint cartilage) even with surgical procedure; may attempt to reattach the flap to the underlying subchondral bone
- Sacrum—remove bone fragment, if impinging on the cauda equina; at this level of the spine, spinal nerves are located in the spinal canal (rather than spinal cord)—these spinal nerves within the spinal canal are known as the “cauda equina”



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs) and pain relievers (known as “analgesics”)—may be used to symptomatically treat degenerative joint disease (progressive and permanent deterioration of joint cartilage) associated with osteochondritis dissecans (abnormal development of bone and cartilage, leading to a flap of cartilage within the joint); does not promote healing of the cartilage flap (thus surgery still is indicated)
- Medications intended to slow the progression of arthritic changes and protect joint cartilage (known as “chondroprotective drugs”), such as polysulfated glycosaminoglycans, glucosamine, and chondroitin sulfate—may help limit cartilage damage and degeneration; may help alleviate pain and inflammation



FOLLOW-UP CARE

PATIENT MONITORING

- Periodic monitoring until pet's skeleton has developed fully and matured—recommended to assess progression to osteochondritis dissecans (abnormal development of bone and cartilage, leading to a flap of cartilage within the joint)
- Yearly examinations—recommended to assess progression of degenerative joint disease (progressive and permanent deterioration of joint cartilage)

PREVENTIONS AND AVOIDANCE

- Discourage breeding of affected dogs
- Do not repeat dam–sire breedings that resulted in affected offspring
- Restricted weight gain and growth in young dogs—may decrease incidence

POSSIBLE COMPLICATIONS

- Degenerative joint disease (progressive and permanent deterioration of joint cartilage)

EXPECTED COURSE AND PROGNOSIS

- Shoulder—good to excellent prognosis for return to full function; minimal osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage) with osteochondrosis and after surgery for osteochondritis dissecans (abnormal development of bone and cartilage, leading to a flap of cartilage within the joint)
- Elbow, stifle, and hock—fair prognosis for osteochondrosis, guarded for osteochondritis dissecans (abnormal development of bone and cartilage, leading to a flap of cartilage within the joint); depends on size of lesion (most important), degenerative joint disease (progressive and permanent deterioration of joint cartilage), and age at diagnosis and treatment; progressive osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage) development, even after surgery
- Sacrum—good after bone fragment removal



KEY POINTS

- Osteochondrosis has a genetic basis
- Degenerative joint disease (progressive and permanent deterioration of joint cartilage) may develop
- Excessive intake of nutrients that promote rapid growth has an influence on the development of osteochondrosis; therefore, restricted weight gain and growth in young dogs may decrease the incidence of osteochondrosis

OSTEOMYELITIS

(INFLAMMATION OF THE BONE AND BONE MARROW)



BASICS

OVERVIEW

- Sudden (acute) or long-term (chronic) inflammation of bone and the associated soft-tissue elements of bone marrow, endosteum (lining of the inner surface of the bone), and periosteum (membrane covering the outer surface of the bone)
- Usually caused by bacteria and rarely by fungi and other microorganisms

GENETICS

- Breeds with inherited immunodeficiency or blood-related diseases

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Breeds with inherited immunodeficiency and blood-related diseases

Mean Age and Range

- Blood-borne infections of the metaphysis (area between the end and the shaft of the bone, where bone growth occurs)—young dogs

Predominant Sex

- Male dogs—for post-traumatic infection; blastomycosis (a fungal infection)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden (acute) postoperative wound infections after orthopedic surgery may mimic signs of sudden (acute) osteomyelitis; may progress to long-term (chronic) disease
- Most patients have long-term (chronic) disease at time of examination and diagnosis
- Episodes of lameness
- Draining tracts
- Persistent ulcers
- Previous trauma
- Fracture or surgery—post-traumatic disease
- Affected vertebrae or intervertebral disks (dogs)—may note hind-limb weakness and difficulty in rising
- Travel to regions where fungal (mycotic) infections are common—fungal infection
- Sudden (acute) blood-borne disease (dogs)—sudden onset of systemic illness; fever (known as “pyrexia”); sluggishness (lethargy); limb pain; local signs of acute inflammation
- Long-term (chronic) condition—usually associated with chronic draining tracts, nonhealing ulcers, pain, secondary loss of muscle mass (known as “muscle atrophy”), and joint stiffness
- Unhealed fractures with infection—may note instability, grating detected with movement (known as “crepitus”), and limb deformity
- Fungal infections—may see limb swelling, lameness, and intermittently draining tracts
- Bone infections of the spine—may cause pain and nervous deficits (such as paralysis)

CAUSES

- Open fracture (that is, a fracture for which the skin is punctured, leaving an open wound to the fracture)
- Traumatic injury
- Surgical repair with metal implants of a closed fracture (that is, a fracture for which the skin was intact, prior to surgery)
- Elective orthopedic surgery
- Prosthetic or artificial joint implant
- Gunshot wound
- Penetrating foreign body
- Bite and claw wounds

- Involvement of bone from soft-tissue infection—inflammation/infection of tissues around and supporting teeth (known as “periodontitis”); inflammation/infection of the nose (known as “rhinitis”); inflammation/infection of the middle ear (known as “otitis media”); inflammation/infection of the nail bed (known as “paronychia”)
- Blood-borne infection
- Staphylococci—cause approximately 50% of bone infections; often single type of bacteria (known as “monomicrobial”) infections
- More than one type of bacteria (known as “polymicrobial”) infection—common; may contain mixtures of aerobic gram-negative bacteria (bacteria that can live and grow in the presence of oxygen); anaerobic bacterial cultures (for bacteria that can live and grow in the absence of oxygen) should be submitted with potential isolates including: *Actinomyces*, *Clostridium*, *Peptostreptococcus*, *Bacteroides*, and *Fusobacterium*
- Fungal infection—*Coccidioides immitis*; *Blastomyces dermatitidis*; *Histoplasma capsulatum*; *Cryptococcus neoformans*; *Aspergillus*

RISK FACTORS

- Open fracture (fracture for which the skin is punctured, leaving an open wound to the fracture) and bone contamination
- Soft-tissue trauma
- Bite and claw wounds
- Migrating foreign body
- Orthopedic surgery
- Prosthetic or artificial joint implant or metal surgical implant (such as bone plate)
- Cortical bone allograft (bone graft transplanted from genetically nonidentical animals of the same species)
- Immunodeficiency
- Methicillin-resistant bacteria populations are increasing in hospitals and veterinary clinics



TREATMENT

HEALTH CARE

- Inpatient—surgical removal of tissue (known as “débridement”), drainage, flushing the wound (known as “irrigation”), and wound management until infection begins to resolve; infected fractures (surgical stabilization)
- Outpatient—long-term antibiotics, administered by mouth
- Depends on severity, location, and degree of associated soft-tissue injury
- Take care to prevent infections by bacterial contamination from other patients in the hospital, so called “hospital-related infections”

ACTIVITY

- Restricted—with any danger of a fracture occurring at the site of weakened bone (known as “pathologic fracture”); with an unhealed fracture

DIET

- No restriction

SURGERY

- Long-term (chronic disease)—surgical removal of tissue (débridement); removal of pieces of dead bone or bone that has become separated from blood supply (known as “sequestra”); establishment of drainage
- Infected stable fracture—leave pre-existing metal surgical implants in place during healing
- Infected unstable fracture—remove metal surgical implants; stabilize fracture by other techniques
- Bone deficits—bone graft
- Localized long-term (chronic) infection—may resolve infection by amputation (tail, digit, limb) or by surgically removing the entire affected area (known as “en bloc resection”)—sternum, thoracic wall, lower jaw [mandible], upper jaw [maxilla]—and primary wound closure
- Remove all metal surgical implants after the fracture has healed



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics—depend on susceptibility of microorganisms; also consider possible toxicity, frequency and route of administration, and expense; most penetrate normal and infected bone well; must be given for 4 to 8 weeks, possibly longer
- Staphylococci (dogs)—usually *Staphylococcus intermedius*, which are resistant to penicillin because of β -lactamase production; highly susceptible to cloxacillin, amoxicillin-clavulanate, cefazolin, and clindamycin
- Anaerobes (bacteria that can live and grow in the absence of oxygen)—more are sensitive to metronidazole and clindamycin
- Aminoglycosides and quinolones (ciprofloxacin and enrofloxacin)—effective against gram-negative aerobic bacteria (bacteria that can live and grow in the presence of oxygen)
- Quinolones—usually used only for infections caused by gram-negative organisms or *Pseudomonas* that are resistant to other antibiotics that can be given by mouth
- Long-term (chronic) disease—continuous local delivery of drugs by antibiotic-impregnated methylmethacrylate beads
- Itraconazole—used to treat fungal infections; given continuously, may control disseminated aspergillosis for up to 2 years
- Identify other antimicrobial drugs by repeating cultures and susceptibility determination if the infection becomes unresponsive to the initial agent



FOLLOW-UP CARE

PATIENT MONITORING

- X-rays—every 4 to 6 weeks; used to monitor bone healing
- Repeat bacterial culture of the bone for suspected persistent infection

POSSIBLE COMPLICATIONS

- Recurrence of osteomyelitis (inflammation/infection of the bone and bone marrow)
- Long-term (chronic) disease—may result in limb deformity, impaired function, fracture disease, or nervous system deficits
- Cancer—rare complication of long-term (chronic) infection of fractures repaired by metal surgical implants

EXPECTED COURSE AND PROGNOSIS

- Sudden (acute) infection and long-term (chronic) bacterial infection of the intervertebral disks and adjacent bone of the spine (known as “diskospondylitis”)—may be cured by 4 to 8 weeks of antibiotics, if bone death (necrosis) is limited and no fracture is present
- Long-term (chronic) disease—resolution with antibiotics alone unlikely; provide appropriate surgical treatment
- Recurrence of long-term (chronic) infection—evident by return of lameness or draining tracts; may occur weeks, months, or years after the last treatment; may require repeated surgical procedures



KEY POINTS

- Treatment is expensive and therapy is of long duration
- Recurrence of long-term (chronic) infection—evident by return of lameness or draining tracts; may occur weeks, months, or years after the last treatment; may require repeated surgical procedures
- Long-term (chronic) disease—resolution with antibiotics alone unlikely; provide appropriate surgical treatment

OSTEOSARCOMA

(BONE CANCER)



BASICS

OVERVIEW

- Most common primary bone tumor in dogs
- “Appendicular” is an adjective relating to the limbs; “axial” is an adjective relating to the head and trunk of the body
- Osteosarcoma typically affects the appendicular skeleton of large- to giant-breed dogs
- Cancerous (malignant) tumor, with spread to the lungs (known as “lung metastases”) in more than 90% of dogs at the time of diagnosis; lung metastases may be microscopic
- Cats—less common; less aggressive biologic behavior than in dogs

GENETICS

- Does not appear to be inherited; although breed susceptibilities do occur
- Breed size and rate of maturity may be more important than breed or family line

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Dogs—large- to giant-breed dogs
- Cats—domestic shorthair

Mean Age and Range

- Dogs—bimodal peak at 2 years and 7 years; reported as young as 6 months of age
- Cats—mean age, 8.5 years; range, 4 to 18 years of age

Predominant Sex

- Dogs—males predominate (1.2:1) in most reports

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on site
- Signs may be subtle
- Swelling, lameness, and pain common
- Other complaints—lack of appetite (inappetence) and sluggishness (lethargy)
- A firm, painful swelling of the affected site common
- Degree of lameness—varies from mild to non-weight bearing
- Fractures occurring at the site of weakened bone (known as “pathologic fractures”) are rare

CAUSES

- Unknown

RISK FACTORS

- Dogs—large- to giant-breed dogs; metallic implants at fracture-repair sites; history of exposure to ionizing radiation
- Dogs—early spay/neuter suggested as a cause in rottweilers
- Cats—unknown



TREATMENT

HEALTH CARE

- Diagnostic evaluation—outpatient
- Surgery and the first chemotherapy treatment—inpatient
- Subsequent chemotherapy—outpatient
- Manage pain, as needed
- Radiation therapy will decrease pain effectively in dogs and cats

ACTIVITY

- Restricted after surgery, until adequate healing has occurred

SURGERY

Appendicular Sites (relating to the limbs)

- Amputation of affected limb—limb amputated at the forequarter (including the scapula and shoulder joint) or hip
- Limb-sparing or salvage therapy—used for osteosarcoma of the distal radius (bone in the lower front leg); available at a limited number of referral hospitals
- Chemotherapy—recommended after either surgical procedure

Axial Sites (relating to the head and trunk of the body)

- Aggressive surgical removal (excision) of the tumor
- Chemotherapy—recommended after surgery

Soft-Tissue Sites (tissues other than bone)

- Aggressive surgical removal (resection) of the tumor
- Chemotherapy recommended after surgery

Metastasectomy (surgical removal of metastasis)

- Surgical removal of metastasis to the lungs (known as “pulmonary metastasectomy”)—has been described; indicated in animals that: 1) had a long disease-free interval after diagnosis; 2) have less than 3 detectable lung nodules; 3) have a lesion doubling time greater than 30 days

Cats

Appendicular Sites (relating to the limbs)

- Amputation of affected limb
- Chemotherapy may not be necessary

Axial Sites (relating to the head and trunk of the body)

- Attempt aggressive surgical excision—depending on site of lesion
- Local recurrence—main reason for treatment failure

Both Species

- Inoperable cancer—radiation therapy offers marked pain relief



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Postsurgical chemotherapy with a platinum-based protocol—current standard of care; chemotherapeutic drugs include cisplatin, carboplatin (use in cats), and doxorubicin
- Palliative medication is intended to improve the animal’s condition and quality of life, it is not a cure for the cancer; these drugs are used to control pain and/or decrease inflammation; options include: aspirin, piroxicam, or other nonsteroidal anti-inflammatory drugs (NSAIDs); acetaminophen with or without codeine, tramadol or a fentanyl patch—not all of these drugs can be used in combination; consult your pet’s veterinarian for the most appropriate pain management for your pet



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor for reduction of bone-marrow activity (known as “myelosuppression”), resulting in low number of red-blood cells, white-blood cells, and/or platelets; should have a complete blood count (CBC) performed 7 to 10 days after chemotherapy

- Take chest X-rays every 2 to 3 months after surgery
- Take X-rays of graft site for cases with limb-sparing or salvage therapy every 2 to 3 months after surgery, because local recurrence is possible after limb salvage

POSSIBLE COMPLICATIONS

- Spread of cancer (metastasis) to lungs, bone, and soft-tissue sites
- Hypertrophic osteopathy (a bone disorder that causes painful swelling of bone and lameness) with spread of cancer to lungs (lung metastases)

EXPECTED COURSE AND PROGNOSIS

- Long-term prognosis is poor; achievable goals should be to relieve discomfort and prolong a good quality of life

Dogs

- Median survival without treatment, with amputation alone, or with palliative radiation therapy alone—approximately 4 months
- Median survival with surgery and chemotherapy—10 months
- Osteosarcoma of the lower jaw (known as “mandibular osteosarcoma”)—less aggressive than other sites; 1-year survival with surgery alone—71% reported

Cats

- Appendicular (involving the limbs)—median survival with surgery: greater than 2 years
- Axial (involving the head and trunk of the body)—median survival with surgery: 5.5 months



KEY POINTS

- The most common primary bone tumor in dogs
- This disease has an aggressive biologic behavior; therapy should be directed at the painful bone tumor (using either surgery or radiation therapy) as well as at metastatic disease (using chemotherapy)
- Long-term prognosis is poor; achievable goals should be to relieve discomfort and prolong a good quality of life

OTITIS EXTERNA AND MEDIA

(INFLAMMATION OF THE OUTER EAR AND MIDDLE EAR)



BASICS

OVERVIEW

- “Otitis externa”—inflammation of the outer ear or external ear canal
- “Otitis media”—inflammation of the middle ear
- The terms are not diagnoses, but rather are descriptions of clinical signs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Pendulous-eared dogs, especially spaniels and retrievers
- Dogs with hair in their external canals—terriers and poodles
- Dogs with narrowing (known as “stenosis”) of the external ear canal—Chinese shar peis

SIGNS/OBSERVED CHANGES in the ANIMAL

- Inflammation of the outer ear (otitis externa)—often secondary to an underlying disease; may see signs of underlying disease
- Inflammation—discharge in the outer ear canal, pain, itchiness (known as “pruritus”) that may lead to rubbing or scratching the ears, and redness of the outer ear (known as “erythema”)
- Infection—discharge in the outer ear canal that contains pus and frequently has a bad odor
- Long-term (chronic) inflammation of the outer ear (otitis externa) in dogs—results in rupture of the ear drum (known as the “tympanic membrane”) in 71% of cases and in inflammation of the middle ear (otitis media) in 82% of cases
- Pain
- Head shaking
- Scratching at the ears
- Redness and swelling of the outer ear canal, leading to narrowing (stenosis) of the canal
- Cats tend to hold the ear down or tilt the head
- Abnormality in which the animal’s sense of balance is altered (known as a “vestibular disorder”) may lead to signs (such as head tilt; short, rapid movements of the eyeball [known as “nystagmus”]; lack of appetite [known as “anorexia”]; wobbly, incoordinated or “drunken” appearing gait or movement [known as “ataxia”]; and infrequent vomiting) that indicate development of inflammation of the middle ear (otitis media) and inflammation of the inner ear (known as “otitis interna”)

CAUSES

Primary Causes

- Parasites (causing inflammation of the outer ear [otitis externa])—ear mites (*Otodectes cynotis*), other mites (*Demodex*, *Sarcoptes* and *Notoedres*), and the spinose ear tick (*Otobius megnini*)
- Hypersensitivities—atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems), food allergy, contact allergy, and generalized (systemic) or local drug reaction
- Foreign bodies—plant awns
- Blockages in the ear canal—tumor or cancer, polyps, enlargement of the glands that secrete ear wax, and accumulation of hair; also may be secondary to other causes of outer ear problems
- Disorder in the normal replacement and shedding of skin cells (known as a “keratinization disorder”) and increased wax production—functional obstruction of the ear canal
- Autoimmune diseases (in which the immune system attacks the body’s own tissues)—frequently affect the ear flap; sometimes affect the external ear canal

Perpetuating Factors

- Secondary bacterial infections—common; *Staphylococcus intermedius* most often cultured from the horizontal ear canal in inflammation of the outer ear (otitis externa); *Pseudomonas*, *Proteus*, *Corynebacterium*, and *E. coli* frequently reported; *Pseudomonas* most often cultured in inflammation of the middle ear (otitis media)

- Infections—often mixed with, or entirely the result of the yeast, *Malassezia pachydermatis*; other yeast (*Candida*) or fungal species are rarely present
- Progressive changes—thickening or enlargement of the tissue of the external ear canal, enlargement of the glands that secrete wax, scar tissue, and cartilage calcification; cause treatment-resistant inflammation of the outer ear (otitis externa); prevent return to a “normal” ear canal even with proper treatment
- Inflammation of the middle ear (otitis media)—can produce signs on its own; can act as a reservoir for disease-causing organisms, leading to recurrent infections

RISK FACTORS

- Abnormal or breed-related conformation of the external ear canal (such as narrowing of the canal, hair in the ear canal, and pendulous ear flaps) restricts proper air flow into the canal
- Excessive moisture (such as from swimming, bathing or frequent ear cleanings with certain ear products) in the ear canal can lead to infection; overzealous client compliance with recommendations for ear cleanings are common
- Reaction to medications applied to the ear directly (known as “topical ear medications”) and irritation and trauma from abrasive cleaning techniques
- Underlying generalized (systemic) diseases produce abnormalities in the ear-canal environment and immune response



TREATMENT

HEALTH CARE

- Outpatient, unless pet has severe vestibular signs (signs due to an abnormality in which the animal’s sense of balance is altered; signs may include head tilt; short, rapid movements of the eyeball [nystagmus]; lack of appetite [anorexia]; wobbly, incoordinated or “drunken” appearing gait or movement [ataxia]; and infrequent vomiting)

ACTIVITY

- No restrictions, unless pet has severe vestibular signs

DIET

- No restrictions, unless a food allergy is suspected

SURGERY

- Indicated when the ear canal is severely narrowed or blocked or when a tumor, cancer or polyp is diagnosed
- Severe, medical treatment-unresponsive inflammation of the middle ear (otitis media) may require surgery to drain the middle ear (procedure known as “bullae osteotomy”) or to remove part of the outer ear (known as “ear ablation”) through the horizontal ear canal



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Systemic Treatment (medications given by mouth, by injection, or by application to the body)

- Antibiotics—useful in severe cases of bacterial infection/inflammation of the outer ear (otitis externa); necessary when the ear drum (tympanic membrane) has ruptured; suggested initial antibiotic choices include cephalexin, enrofloxacin, or clindamycin; antibiotic-resistant infections require bacterial culture and sensitivity of the discharge in the ear to determine antibiotic selection
- Medications to treat yeast or fungal infections (known as “antifungals”)—use with overwhelming yeast or fungal infection; example is ketoconazole
- Steroids—reduce swelling and pain; reduce wax production; anti-inflammatory dosages of prednisone; use sparingly and for short duration
- Selamectin—(Revolution®) for ear mites, applied onto body every 2 weeks for 3 applications

Topical Treatment (medications applied to the ear canal directly)

- Topical therapy is very important for resolution and control of inflammation of the outer ear (otitis externa)
- First, completely clean the external ear canal of debris; complete flushing under general anesthesia may be necessary, especially for uncooperative patients or severe cases, including those with inflammation of the middle ear (otitis media)

- Second, thoroughly clean the ear daily or every other day during initial therapy; then every 3 to 7 days once signs resolve
- Finally, apply appropriate topical medications frequently and in sufficient quantity to completely treat the entire ear canal
- Combination ointments are not recommended because they often accumulate in the ear canal and may perpetuate the condition
- Suggested topical medications include antibiotics (such as gentamicin) or antifungal drops (such as miconazole) for yeast or fungal infections, with or without steroids
- Commercial ear cleansers with compounds to soften and breakdown the ear wax (known as “cerumenolytics”), compounds to slow the growth of bacteria (known as “antiseptics”), and compounds to decrease secretions and reduce moisture (known as “astringents”); your pet’s veterinarian will recommend the appropriate ear cleanser for your pet and will provide directions for use
- Cerumenolytics—dioctyl sodium sulfosuccinate or carbamide peroxide; emulsify waxes, facilitating removal of wax and debris
- Antiseptics—acetic acid or chlorhexidine gluconate; reduce or eliminate infectious organisms
- Astringents—isopropyl alcohol, boric acid, or salicylic acid; reduce moisture
- Antibiotics, antifungals, and/or parasiticides—use when presence of organism(s) has been confirmed
- Ivermectin 0.01% (Acarexx® Otic Suspension)—FDA-labeled to treat ear mites (*Otodectes cynotis*)
- Resistance to medications—perform a bacterial culture and sensitivity of the ear discharge
- Generally, ingredients of topical medications should be limited to those needed to treat a specific infection (that is, medications containing antibiotics should be used only for bacterial infections)



FOLLOW-UP CARE

PATIENT MONITORING

- Follow-up examinations and evaluations of ear discharge can assist in monitoring infection

PREVENTIONS AND AVOIDANCE

- Routine ear cleaning at home, as directed by your pet’s veterinarian
- Control of underlying diseases

POSSIBLE COMPLICATIONS

- Uncontrolled inflammation of the outer ear (otitis externa) can lead to inflammation of the middle ear (otitis media); deafness; vestibular disease (abnormality in which the animal’s sense of balance is altered); inflammation of the tissues under the skin that tends to spread (known as “cellulitis”); facial nerve paralysis; progression to inflammation of the inner ear (known as “otitis interna”); and rarely inflammation of the brain and its surrounding membranes (known as “meningoencephalitis”)

EXPECTED COURSE AND PROGNOSIS

- Inflammation of the outer ear (otitis externa)—with proper therapy, most cases resolve in 3 to 4 weeks; failure to correct underlying primary cause often results in recurrence
- Perpetuating factors (such as narrowing of the ear canal and calcification of the cartilage of the ear) will not resolve and may result in recurrence
- Inflammation of the middle ear (otitis media)—may take 6+ weeks of systemic antibiotics until all signs have resolved and the ear drum (tympanic membrane) has healed



KEY POINTS

- The proper method for cleaning ears is very important; talk to your pet’s veterinarian so you understand the procedure and frequency of ear cleaning

OTITIS MEDIA AND INTERNA (INFLAMMATION OF THE MIDDLE EAR AND INNER EAR)



BASICS

OVERVIEW

- Inflammation of the middle ear (known as “otitis media”) and inner ear (known as “otitis interna”), most commonly caused by bacterial infection

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Cocker spaniels and other long-eared breeds
- Poodles with long-term (chronic) inflammation of the ears (known as “otitis”) or the throat (known as “pharyngitis”) associated with dental disease

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on severity and extent of the infection; range from no signs to those related to middle ear discomfort and nervous system involvement
- Pain when opening the mouth; reluctance to chew; shaking the head; pawing at the affected ear
- Head tilt
- Pet may lean, veer, or roll toward the side or direction of the affected ear
- Animal’s sense of balance may be altered (known as “vestibular deficits”)—persistent, transient or episodic
- Involvement of both ears—wide movements of the head, swinging back and forth; wobbly or incoordinated movement of the body (known as “truncal ataxia”), and deafness
- Vomiting and nausea—may occur during the sudden (acute) phase
- Facial nerve damage—the “facial nerve” goes to the muscles of the face, where it controls movement and expression, as well as to the tongue, where it is involved in the sensation of taste; signs of facial nerve damage include saliva and food dropping from the corner of the mouth; inability to blink; discharge from the eye; weakness (known as “paresis”) or paralysis of the affected ear and the eyelids, lips, and nostrils on the side of the affected ear; may have reduced tear production; with long-term (chronic) facial nerve paralysis, the face may contract or twist toward the affected side, caused by development of scar tissue in the muscles
- Unequal size of the pupils (known as “anisocoria”), the pupil is smaller on the side of the affected ear, protrusion of the third eyelid, the eyeball is withdrawn into the socket (known as “enophthalmos”) and the upper eyelid droops (known as “ptosis”)—these signs are known as “Horner’s syndrome”
- Evidence of redness of the ear, discharge, and thick and narrowed external ear canals indicates inflammation of the outer ear (known as “otitis externa”)
- Gray, dull, opaque, and bulging eardrum (known as the “tympanic membrane”), observed during examination using an otoscope to look down into the ear canal—indicates some type of fluid build-up in the middle ear
- Dental tartar, inflammation of the gums (known as “gingivitis”), of the tonsils (known as “tonsillitis”), or the throat (pharyngitis)—may be present and associated with inflammation of the middle ear (otitis media) and inner ear (otitis interna)
- Enlargement of the mandibular lymph node on the side of the ear inflammation (known as “mandibular lymphadenopathy”)—may occur with severe infections
- Superficial loss of tissue on the surface of the cornea, the clear outer layer of the front of the eye (known as a “corneal ulcer”)—may be caused by inability to blink or a dry eye
- Signs associated with damage to nervous system structures depend on the severity and location
- Pet may be reluctant to move and may stay in a crouched posture with wide movements of the head, swinging back and forth
- Short, rapid movements of the eyeball (known as “nystagmus”)

CAUSES

- Bacteria—primary disease-causing agents
- Yeast (*Malassezia*, *Candida*) and fungus (*Aspergillus*)—possible disease-causing agents
- Mites—increase likelihood of secondary bacterial infections

- Disease involving only one ear—foreign bodies, trauma, polyps, and tumors (such as fibromas, squamous cell carcinoma, ceruminous gland carcinoma, and primary bone tumors)

RISK FACTORS

- Inflammatory masses that develop from the middle ear or eustachian tube (known as “nasopharyngeal polyps”) and tumors or cancer of the inner, middle, or outer ear—may increase susceptibility to bacterial infection
- Vigorous ear flush
- Ear-cleaning solutions (such as chlorhexidine)—may be irritating to the middle and inner ear; avoid if the ear drum (tympanic membrane) is ruptured
- Inhalant anesthesia and traveling by airplane—change middle-ear pressures



TREATMENT

HEALTH CARE

- Inpatient—severe debilitating infection; nervous system signs
- Fluid therapy—if pet is unable to eat or drink, owing to nausea or vomiting and/or disorientation
- Coexistent inflammation of the outer ear (otitis externa)—bacterial culture and sensitivity testing; clean the ear; use warm normal saline if the ear drum (tympanic membrane) is ruptured; if a cleaning solution is used, follow with a thorough flush with normal saline; carefully dry the ear canal (drying products [known as “astringents”], such as Otic Domeboro® solution or boric acid, can be effective)

ACTIVITY

- Restrict activity in pets with substantial alteration of sense of balance (vestibular disorder) to avoid injury

DIET

- Vomiting from alteration of sense of balance (vestibular disorder)—withhold food and water for 12 to 24 hours
- Severe disorientation—hand feed and water small amounts frequently; elevate head to avoid aspiration pneumonia

SURGERY

- Reserve surgery for patients that have relapsing inflammation of the middle and inner ear (otitis media/interna), that are not responding to medical treatment or that are deteriorating
- Do not rely on severity of nervous system signs as an indication for surgical intervention; reserve surgery for patients with evidence of fluid build-up in the middle ear; infection/inflammation of the bone (known as “osteomyelitis”) surrounding the ear that is not responsive to medical management; and presence of inflammatory masses that develop from the middle ear or eustachian tube (nasopharyngeal polyps) or tumors/cancer
- Surgery may be indicated in some cases of inflammation of the middle ear (otitis media) to drain the middle ear cavity (procedure known as “bullae osteotomy”)
- Surgical removal of part of the outer ear (known as “ear ablation”) through the horizontal ear canal—indicated when inflammation of the middle ear (otitis media) is associated with recurrent inflammation of the outer ear (otitis externa) or tumors/cancer
- Obtains samples at time of surgery for microscopic examination of abnormal tissue and for bacterial culture and sensitivity of fluid in the middle ear



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Topical (applied directly into the ear) antibiotic solutions—chloramphenicol or a triple antibiotic preparation; or ofloxacin otic solution (Floxin® Otic)—dogs and cats
- Antibiotics—broad-spectrum antibiotics administered by mouth or injection; long-term treatment (6 to 8 weeks, if presumptive diagnosis); select antibiotics on basis of bacterial culture and sensitivity testing, if available
- Amoxicillin/clavulanic acid (Clavamox®)—good first choice antibiotic
- Fluoroquinolone or third generation cephalosporin antibiotics are good second choice alternatives or can be used in combination, if bacterial culture and sensitivity test results are unavailable; examples include enrofloxacin (Baytril®), marbofloxacin (Zeniquin®), or

cefepodoxime (Simplicef®)

- Clindamycin (Cleocin®) may be administered, if anaerobes are suspected; “anaerobes” are bacteria that can live and grow in the absence of oxygen



FOLLOW-UP CARE

PATIENT MONITORING

- Evaluate after 10 to 14 days, or sooner if the patient’s condition is deteriorating

PREVENTIONS AND AVOIDANCE

- Routine ear cleaning—may reduce chances of infection of the middle ear and inner ear
- Routine professional teeth cleaning, with the animal under anesthesia (known as a “dental prophylaxis”)—may reduce chances of infection of the middle and inner ear

POSSIBLE COMPLICATIONS

- Altered sense of balance (vestibular disorder), facial nerve damage or Horner’s syndrome (condition in which one pupil is small or constricted, the eyelid droops, and the eyeball is withdrawn into the socket); the “facial nerve” goes to the muscles of the face and controls movement and expression, as well as to the tongue, where it is involved in the sensation of taste; signs of facial nerve damage include weakness (known as “paresis”) or paralysis of the affected ear and the eyelids, lips, and nostrils on the side of the affected ear
- Severe middle/inner ear infections—infection may spread to the brain stem
- Infection/inflammation (osteomyelitis) of the bone of the skull around the ear and fluid build-up in the middle ear cavity—common sequela to severe, long-term (chronic) inflammation of the outer ear (otitis externa)
- Surgery to drain the middle ear (procedure is a bulla osteotomy)—postoperative complications include Horner’s syndrome (condition in which one pupil is small or constricted, the eyelid droops, and the eyeball is withdrawn into the socket), facial paralysis, and onset or worsening of an altered sense of balance (vestibular disorder), or deafness with infections involving both ears

EXPECTED COURSE AND PROGNOSIS

- Inflammation of the middle ear (otitis media) and inner ear (otitis interna)—usually responsive to medical management; 2- to 4-month course of antibiotics should be considered to avoid relapse
- Signs associated with an altered sense of balance (vestibular disorder)—improvement in 2 to 6 weeks; more rapid in small dogs and in cats



KEY POINTS

- Most bacterial infections of the middle ear and inner ear resolve with an early, aggressive course of long-term, broad-spectrum antibiotics
- Relapsing signs may occur
- Surgical drainage of the middle ear may be necessary

PAIN (ACUTE, CHRONIC, AND POSTOPERATIVE)



BASICS

OVERVIEW

- Pain is an unpleasant sensory or emotional experience associated with actual or potential tissue damage
- The inability to communicate in no way negates the possibility that an animal is experiencing pain and is in need of appropriate pain-relieving treatment
- “Acute” is the medical term for “sudden;” “chronic” is the medical term for “long-term;” and “postoperative” is the medical term for “following surgery”

GENETICS

- Evidence suggests that age, sex, breeding strain, and species can alter responses to harmful or injurious stimuli (known as “noxious stimuli”)
- Recently, genes have been described that modify individual mouse behavioral responses to harmful or injurious stimuli (noxious stimuli)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Behavioral signs of pain and distress vary considerably among individual animals
- Experience, environment, age, species, and other factors can modify the intensity of the reaction to harmful or injurious stimuli (noxious stimuli)
- Most obvious clinical signs of distress in the dog and cat—vocalization; agitation; abnormal posture or gait; thrashing; being overly sensitive to pain or touch (known as “hyperesthesia”) or extremely sensitive to painful stimuli (known as “hyperalgesia”); and being extremely sensitive to stimuli that normally would not cause discomfort or pain, but the animal responds with a painful response (known as “allodynia”)
- More subtle signs include trembling, depression, reduced appetite, stupor, and biting
- Rapid breathing (known as “tachypnea”), rapid heart rate (known as “tachycardia”), dilated pupils (known as “mydriasis”), and increased blood pressure (known as “hypertension”)—associated with the stress response; may accompany pain, but are nonspecific signs that may be seen with many conditions
- Clinical signs associated with long-term (chronic) pain may be very subtle or difficult to evaluate, since the animal may compensate for the pain; chronic pain often is associated with decreased activity, lameness, or depression

CAUSES

- Pain can be caused by tissue disruption associated with trauma or surgery, but it also is caused by long-term (chronic) degenerative changes, such as osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage)
- Pain that outlives the initial tissue damage may indicate altered nervous system processing

RISK FACTORS

- Trauma
- Surgery
- Pain intensity may not always correlate with the degree of tissue damage; however, more invasive soft-tissue and orthopedic surgical procedures likely are associated with a greater intensity of pain than less invasive surgical procedures



TREATMENT

HEALTH CARE

- Medications to decrease pain (known as “analgesics”) and to produce a loss of ability to perceive pain (known as “anesthetics”); drug selection depends on species, pain intensity, and underlying cause of pain

- Treat the underlying cause at the same time, if possible
- General good management and nursing practices to make the pet comfortable (for example, use of padded bedding)
- Non-pharmacologic treatments, including bandaging and hydrotherapy (which may be achieved with a whirlpool bath) may be appropriate
- Acupuncture and chiropractic manipulation may be useful additional treatment options for certain conditions
- If the patient's quality of life is not acceptable, euthanasia may be the most humane option

ACTIVITY

- Cage rest, limited activity, or physical therapy may be useful for certain types of pain

DIET

- Dietary changes to help treat the underlying condition (such as weight reduction to help treat hip dysplasia) may be beneficial
- Many supplements and nutraceuticals are marketed commercially; they claim to have beneficial effects on joint cartilage
- Commercial veterinary diets are marketed specifically for dogs with osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage)

SURGERY

- Surgical treatment of the underlying condition causing pain may be the best treatment
- Surgical disruption of nerves (known as "neurectomy") to halt pain transmission—not always associated with positive results; may result in worsening of the painful condition



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Opioids, alone or in combination with other classes of drugs (such as sedative/tranquilizers or nonsteroidal anti-inflammatory drugs [NSAIDs]) are used widely for the management of sudden (acute) postoperative pain; examples of opioids include morphine, hydromorphone, and fentanyl for moderate to severe pain; buprenorphine and butorphanol for mild to moderate pain
- Nonsteroidal anti-inflammatory drugs (NSAIDs) are used most commonly for the long-term (chronic) treatment of painful conditions in dogs; newer NSAIDs have improved safety when administered over a prolonged period, but gastrointestinal and kidney side effects are still possible—talk to your pet's veterinarian about possible side effects
- The safety and effectiveness of most nonsteroidal anti-inflammatory drugs (NSAIDs) have not been well demonstrated in cats, and their long-term use in cats is limited
- Treatment of pain related to disorders of the nervous system or to altered nervous system processing (known as "neuropathic pain") is challenging; neuropathic pain does not always respond well to traditional pain relievers (analgesics), such as opioids and nonsteroidal anti-inflammatory drugs (NSAIDs), although these drugs usually are tried initially; if the animal does not respond to analgesics, other medications (such as tricyclic antidepressants or antiepileptic drugs) and other alternative (complementary) therapies may be effective
- Non-traditional medical treatments are common, but should be evaluated for safety and effectiveness before use



FOLLOW-UP CARE

PATIENT MONITORING

- Frequent evaluation of the effectiveness of medications designed to decrease pain (analgesics) should be performed in order to ensure the humane care of animals
- Pets receiving long-term (chronic) medications to decrease pain (analgesics), especially nonsteroidal anti-inflammatory drugs (NSAIDs), should be evaluated periodically to monitor gastrointestinal, liver, and kidney function
- Careful follow-up and monitoring are important, especially when treating animals following trauma and in the immediate period following surgery

PREVENTIONS AND AVOIDANCE

- Although some degree of pain is usually an unavoidable consequence of trauma or surgery, when possible, the administration of medications to decrease pain (analgesics) early in treatment or before the animal experiences pain may provide better control of the

pain

- Use of anesthetic techniques incorporating premedications to decrease pain (known as “analgesic premedication”)

POSSIBLE COMPLICATIONS

- Usually minimal

EXPECTED COURSE AND PROGNOSIS

- Sudden (acute) pain associated with trauma or surgery usually resolves with tissue healing
- Opioids may be most effective for the 12 to 24 hours following surgery, whereas nonsteroidal anti-inflammatory drugs (NSAIDs) may be better after that period
- Some nonsteroidal anti-inflammatory drugs (NSAIDs) are effective medications to decrease pain (analgesics), when given immediately after surgery
- When pain signs persist beyond the normal course of a few days to weeks, suspect persistent disease, injury, or central nervous system changes; if pain signs persist, your veterinarian may consult an anesthesiologist or a specialist trained in pain management for suggestions about appropriate therapy



KEY POINTS

- Understand what to expect and to look for when your pet is receiving medications to relieve pain (analgesics)
- The veterinarian should explain the effects of the prescribed medication as well as side effects; if you have any questions, ask your pet's veterinarian
- Effectiveness of medications to relieve pain (analgesics) varies and several drugs may need to be tried before an effective treatment is found for your individual pet
- You know your pet better than anyone; talk to your veterinarian about participating in evaluation of your pet for pain, especially long-term (chronic) pain

NECK AND BACK PAIN



BASICS

OVERVIEW

- Discomfort along the spine or vertebral column; discomfort may involve the spinal cord, spinal nerves, bones, and/or muscles along the spine
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae
- Each disk is composed of a central gel-like area, known as the “nucleus pulposus,” and an outer fibrous ring, known as the “annulus fibrosis”
- Degeneration of the intervertebral disks causes protrusion or extrusion of disk material into the spinal canal; the protruded or extruded disk material causes spinal-cord compression (known as “myelopathy”) and/or nerve-root compression (known as “radiculopathy”)
- Protrusion is defined as the disk bulging into the spinal canal with the fibrous ring of the disk being intact; extrusion is defined as the center or nucleus of the disk being forced out of its normal position into the spinal canal with the fibrous ring of the disk being ruptured
- Two types of protrusion/extrusion have been reported in dogs: sudden (acute) disk herniation (“slipped disk”) is Hansen type I and long-term (chronic) disk herniation is Hansen type II; Hansen type I involves degeneration of the center or nucleus of the disk with rupture of the fibrous ring and resulting movement of the center into the spinal cord (extrusion) while Hansen type II involves degeneration of the disk, followed by bulging of the disk into the spinal cord with the fibrous ring remaining intact (protrusion)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions and Ages

- Intervertebral disk disease—dogs: Hansen type I usually develops at 3 to 8 years of age, occasionally outside this range; Hansen type II is more common in large-breed, older dogs in the low lumbar area; more frequently recognized in low lumbar area in cats
- “Wobbler” syndrome (condition affecting the cervical spine, in which the spinal cord is compressed; may involve the intervertebral disks or abnormal bones [vertebrae])—large-breed dogs; more often in middle-aged to older Doberman pinschers (disk related) and young Great Danes (abnormal formation and/or abnormal movement of the bones [vertebrae] in the neck)
- Dislocation of the joint between the first and second cervical vertebra (condition known as “atlantoaxial luxation”) and partial dislocation of the joint between the first and second cervical vertebra (condition known as “atlantoaxial subluxation”)—most often occurs in young to middle-aged miniature breeds; any breed or age may be affected, if trauma induced
- Steroid-responsive inflammation of the membranes covering the brain and spinal cord (membranes known as “meninges;” condition known as “meningitis”) and inflammation of the arteries (known as “arteritis”)—dogs less than 2 years of age; Bernese mountain dog, boxer, beagle, Nova Scotia duck tolling retriever, German shorthaired pointer
- Bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition known as “diskospondylitis”)—dogs; intact breeding animals susceptible to *Brucella* diskospondylitis
- Musculoskeletal trauma—any age or breed

SIGNS/OBSERVED CHANGES in the ANIMAL

- Perceived discomfort of the animal (such as reluctance to get up or lie down, reluctance to go up or down stairs, difficulty squatting to urinate or defecate, difficulty getting into vehicles)
- Head down posture—neck pain
- Reluctance to move head in various directions—stiff neck
- Arched back—neck or back pain
- Pain on feeling the epaxial muscles (muscles along the spine)
- Rigidity of the epaxial muscle (muscles along the spine)
- Warmth or heat detected in the epaxial muscles (muscles along the spine)
- Guarded posture
- Reluctance to walk—guarded short stride

- Low-grade fever—primarily in patients with involvement of the membranes covering the brain and spinal cord (meninges) or bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (diskospondylitis)

CAUSES

Epaxial Muscles (muscles along the spine)

- Inflammation of the muscles secondary to trauma (known as “traumatic myositis”)
- Disorder following exercise that leads to injury and destruction of skeletal muscle tissue (known as “exertional rhabdomyolysis”)
- Muscle cancer—rhabdomyosarcoma
- Infectious inflammation of the muscles (infectious myositis)—parasitic, bacterial, protozoal
- Immune-mediated inflammation of the muscles (immune-mediated myositis)
- Foreign-body inflammation of the muscles (foreign-body myositis)—grass-awn migration

Backbone (Vertebra) and Associated Structures

- Intervertebral disk disease
- Bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (diskospondylitis)
- Osteoarthritis (form of joint inflammation [arthritis] characterized by chronic deterioration or degeneration of the joint cartilage) of the articular facets (surfaces of the backbone [vertebra] where it joins together with another backbone)
- Unstable structural abnormalities of the backbones—hemivertebra (incomplete development of one side of a vertebra) and dislocation of the joint between the first and second cervical vertebra (atlantoaxial luxation) or partial dislocation of the joint between the first and second cervical vertebra (atlantoaxial subluxation)
- Cancer of the backbone (vertebra)—osteosarcoma, chondrosarcoma, multiple myeloma, and cancer that has spread to the spine (known as “metastatic cancer”)
- Infection/inflammation of the backbone (vertebra; condition known as “vertebral osteomyelitis”)
- Fracture
- Dislocation (known as “luxation”) and partial dislocation (known as “subluxation”)
- Abnormal formation and abnormal movement of the backbones (vertebrae)

Spinal Nerves

- Entrapment of the spinal nerve by intervertebral disk herniation
- Tumors or cancer—neurofibroma and neurofibrosarcoma
- Traumatic entrapment, tearing, or laceration of the spinal nerves
- Inflammation of the nerves (known as “neuritis”)—viral, bacterial, and parasitic
- Compression or inflammation of spinal nerves at the point where they enter the spine or vertebral column

Meninges (membranes covering the brain and spinal cord)

- Tumors or cancer—meningioma and cancer that has spread to the meninges (metastatic cancer)
- Inflammation of the meninges (known as “meningitis”)—bacterial, viral, parasitic, protozoal, rickettsial, of immune-mediated disease or of unknown cause (so called “idiopathic disease”)

RISK FACTORS

- Trauma
- Breeds with “normal” short, bowed legs (known as “chondrodysplastic breeds”)
- Very active animal—for example, asked to jump a lot (such as working police dogs)
- Previous diagnosis of cancer



TREATMENT

HEALTH CARE

- Varies widely according to the nature and extent of the lesion and tissues involved
- The cause of the neck or back pain should be diagnosed before symptomatic treatment is started
- Inpatient versus outpatient—depends on severity of disease
- Acupuncture—may alleviate neck/back pain

ACTIVITY

- Depends on underlying cause of neck or back pain

SURGERY

- Surgical intervention—may be indicated for intervertebral disk herniation, trauma, congenital (present at birth) spinal abnormalities, and tumors/cancer; a foreign body may require surgical removal or drainage to treat an associated abscess



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Epaxial Muscles (muscles along the spine)

- Antibiotics—for infection; depends on the causative agent
- Steroids—may be required; depends on the diagnosis
- Chemotherapy or radiation therapy—for cancer; depends on tumor type

Backbone (Vertebra) and Associated Structures

- Steroids—indicated in some causes (such as intervertebral disk herniation, other spinal cord trauma) and not indicated in other causes (such as infectious disease); if at all possible, a diagnosis should be established before initiating steroid therapy
- Antibiotics or antifungal medications—indicated when a specific organism can be identified or is suspected (such as with bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine [diskospondylitis])
- Chemotherapy and radiation therapy—depends on tumor type

Spinal Nerves

- Steroids—useful for trauma, inflammation, and nerve compression; may help some patients with tumors or cancer

Meninges (membranes covering the brain and spinal cord)

- Antibiotics—indicated when a specific organism can be identified or is suspected; chose antibiotics that cross the blood–brain barrier
- Steroids—if at all possible, a diagnosis should be established before initiating steroid therapy; drug of choice for immune-mediated or steroid-responsive diseases
- Nonsteroidal anti-inflammatory drugs (NSAIDs)—may alleviate musculoskeletal pain; limited results against nervous system-related pain
- Glycosaminoglycan (such as chondroitin)—may decrease musculoskeletal pain
- Methocarbamol—used for muscle relaxation
- Benzodiazepines (such as diazepam)—muscle relaxation and antianxiety effects
- Phenylbutazone—may alleviate musculoskeletal pain; ineffective against nervous system-related pain
- Fentanyl patches—to relieve pain
- Other pain relievers (known as “analgesics”), such as tramadol, butorphanol, buprenorphine, morphine sulfate



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor response to treatment closely and make adjustments as necessary
- Watch for signs of inflammation of the stomach and intestines (known as “gastroenteritis”) and the bladder (known as “cystitis”)

POSSIBLE COMPLICATIONS

Epaxial Muscles (muscles along the spine)

- Abscess
- Long-term (chronic) pain
- Development of scar tissue (fibrous replacement) in place of muscle fibers, causing long-term (chronic) pain and immobility

Backbone (Vertebra) and Associated Structures

- Frequent recurrence in patients with intervertebral disk disease that receive medical management only
- Permanent paralysis or dysfunction

- Lack of control of urination (known as “urinary incontinence”) and bowel movements (known as “fecal incontinence”)
- Long-term (chronic) pain
- Spread of disease to adjacent tissues

Spinal Nerves

- Permanent nervous system deficit or dysfunction
- Long-term (chronic) pain

Meninges (membranes covering the brain and spinal cord)

- Involvement of spinal cord and brain tissue

EXPECTED COURSE AND PROGNOSIS

- Depend on cause of neck and/or back pain



KEY POINTS

- Discomfort along the spine or vertebral column; discomfort may involve the spinal cord, spinal nerves, bones, and/or muscles along the spine
- Perceived discomfort of the animal (such as reluctance to get up or lie down, reluctance to go up or down stairs, difficulty squatting to urinate or defecate, difficulty getting into vehicles)
- The cause of the neck or back pain should be diagnosed before symptomatic treatment is started

PANCREATITIS

(INFLAMMATION OF THE PANCREAS)



BASICS

OVERVIEW

- Inflammation of the pancreas
- Sudden (acute) pancreatitis—inflammation of the pancreas that occurs abruptly, with little or no permanent damage to the pancreas
- Long-term (chronic) pancreatitis—continuing inflammation of the pancreas that is accompanied by irreversible damage to the pancreas
- “Edematous pancreatitis” is characterized by fluid build-up in the interstitium (small spaces between tissues or parts of the pancreas) and mild inflammation with neutrophils and lymphocytes (two types of white-blood cells); the animal generally recovers rapidly
- “Necrotizing pancreatitis” is inflammation of the pancreas characterized by bleeding (hemorrhage) and areas of death of tissues (known as “necrosis,” thus the name “necrotizing pancreatitis”); it usually is a severe and prolonged disease and many affected animals die

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Miniature schnauzer
- Miniature poodle
- Cocker spaniel
- Siamese (cat)

Mean Age and Range

- Sudden (acute) pancreatitis is most common in middle-aged and old dogs (over 7 years of age); mean age at presentation is 6.5 years
- Mean age for sudden (acute) pancreatitis in cats is 7.3 years

Predominant Sex

- Female—dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Dogs—gastrointestinal tract signs (such as vomiting, diarrhea)
- Cats—vague, non-specific signs that generally do not localize problem to the pancreas
- Sluggishness (lethargy), depression, lack of appetite (anorexia)—common in dogs and cats
- Vomiting—common in dogs, less common in cats
- Diarrhea—more frequently seen in dogs than in cats
- Weight loss—common in cats
- Dogs may exhibit abdominal pain
- Yellowish discoloration to gums and moist tissues of the body (known as “jaundice” or “icterus”)—common in dogs and cats
- Dehydration—common; due to gastrointestinal losses of fluid
- Mass lesions may be felt during physical examination in both dogs and cats.
- Fever—common in dogs; both fever and low body temperature (known as “hypothermia”) reported in cats
- Less common systemic abnormalities include severe breathing difficulties (known as “respiratory distress”), bleeding disorders, and irregular heart beats (known as “cardiac arrhythmias”)

CAUSES

- Usually unknown; possibilities include the following:
 - Nutritional factors (such as an increase in lipoprotein [complexes of lipid and protein] concentration in the blood [known as “hyperlipoproteinemia”])
 - Pancreatic trauma or lack of blood flow (known as “ischemia”) to the pancreas
 - Duodenal reflux (a condition in which contents in the upper small intestine [duodenum] move backward)

- Drugs or toxins
- Pancreatic duct blockage or obstruction
- High levels of calcium in the blood (known as “hypercalcemia”)
- Infectious diseases—toxoplasmosis, feline infectious peritonitis (FIP)
- Extension of inflammation from the liver and bile duct system or intestines in the cat

RISK FACTORS

- Breed—dog: miniature schnauzer, miniature poodle, cocker spaniel; cat: Siamese
- Obesity in dogs
- Another disease (such as sugar diabetes [diabetes mellitus]; increased levels of steroids produced by the adrenal glands [known as “hyperadrenocorticism” or “Cushing’s disease”]; long-term [chronic] kidney failure, and cancer) in dogs
- Recent administration of certain drugs
- Liver (hepatic) or gastrointestinal tract inflammation in cats



TREATMENT

HEALTH CARE

- Inpatient medical management
- Aggressive intravenous (IV) fluid therapy
- Fluid therapy goals—correct low circulating blood volume (known as “hypovolemia”) and maintain pancreatic circulation
- A balanced electrolyte solution (such as lactated Ringer’s solution [LRS]) is the first-choice for providing hydration
- May need colloids; colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch
- Following replacement of fluid deficits, give additional fluids to match maintenance requirements and ongoing losses
- Potassium chloride (KCl) supplementation usually needed, because potassium is lost from the body in the vomit

ACTIVITY

- Restrict

DIET

- Continue to feed by mouth, unless vomiting is difficult to control; feeding maintains the integrity of the intestinal lining and minimizes bacterial invasion from the intestines and into the body
- Animals with intermittent vomiting should be treated with drugs to control nausea and vomiting (known as “antiemetics”), such as metoclopramide or phenothiazines
- Tube feeding into the jejunum (the middle section of the small intestine) allows feeding into the intestines (known as “enteral feeding”), while allowing the pancreas to rest
- Withhold all food and water by mouth (known as “NPO”) in animals with persistent vomiting; when no vomiting has occurred for 24 to 48 hours, offer small volumes of water; if tolerated, begin small, frequent feedings of a carbohydrate (such as boiled rice); gradually introduce a protein source of high biologic value (such as cottage cheese or lean meat)
- Avoid high-protein and high-fat diets
- Patients needing extended time without food and water by mouth (NPO) may require tube feeding into the jejunum or intravenous feeding (known as “total parenteral nutrition”)

SURGERY

- May need surgery to remove localized accumulations of fluid (known as “pseudocysts”), abscesses, or areas of dead (necrotic) tissue seen with necrotizing pancreatitis (inflammation of the pancreas characterized by bleeding and areas of death of tissues)
- May need surgical exploration of the abdomen and biopsy of the pancreas to confirm pancreatitis and/or to rule out other diseases not involving the pancreas
- Bile-duct blockage outside of the liver (known as “extrahepatic biliary obstruction”) from pancreatitis requires surgical correction



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids are indicated only for the treatment of shock
- Drugs that act on the vomiting center of the brain to control nausea and vomiting (known as “centrally acting antiemetics”) are indicated with vomiting that is difficult to control—metoclopramide, chlorpromazine, or prochlorperazine
- Antibiotics, if evidence of sepsis (presence of pus-forming bacteria and their poisons in the blood or tissues)—penicillin G, ampicillin, and enrofloxacin
- Pain relievers (analgesics) to relieve abdominal pain, such as buprenorphine



FOLLOW-UP CARE

PATIENT MONITORING

- Evaluate hydration status closely during first 24 hours of therapy; twice daily check physical examination; body weight; packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood) and total solids (a quick laboratory test that provides general information on the level of protein in the fluid portion of the blood); and blood urea nitrogen (BUN) and urine output to monitor the kidneys and hydration status
- Evaluate the effectiveness of fluid therapy after 24 hours, and adjust flow rates and fluid composition accordingly; repeat blood tests (serum biochemistries) to assess electrolyte/acid–base status
- Repeat plasma enzyme concentrations (pancreatic-lipase immunoreactivity [PLI] assay, a test that determines the levels of lipase, a pancreatic enzyme) after 7 days, to evaluate the status of the inflammation of the pancreas
- Watch closely for complications involving a variety of organ systems; perform appropriate diagnostic tests as needed
- Gradually taper fluids down to maintenance requirements, if possible
- Maintain feeding by mouth or into the jejunum (enteral nutrition)
- Reassess and correct on-going low serum cobalamin (Vitamin B12) concentrations

PREVENTIONS AND AVOIDANCE

- Weight reduction, if obese
- Avoid high-fat diets
- Avoid drugs that may increase the risk of inflammation of the pancreas (pancreatitis)

POSSIBLE COMPLICATIONS

- Failed response to supportive therapy
- Life-threatening associated conditions

EXPECTED COURSE AND PROGNOSIS

- Good for most patients with edematous pancreatitis (inflammation of the pancreas characterized by fluid build-up in the interstitium and mild inflammation with neutrophils and lymphocytes [two types of white-blood cells]); these patients usually respond to appropriate symptomatic therapy
- More guarded to poor for patients with necrotizing pancreatitis (inflammation of the pancreas characterized by bleeding [hemorrhage] and areas of death of tissues [necrosis]) and systemic conditions



KEY POINTS

- Sudden (acute) pancreatitis—inflammation of the pancreas that occurs abruptly, with little or no permanent damage to the pancreas
- Long-term (chronic) pancreatitis—continuing inflammation of the pancreas that is accompanied by irreversible damage to the pancreas

- Need for extended hospitalization
- Diagnosis and treatment can be expensive
- Possible complications include lack of response to supportive therapy and life-threatening conditions

PANCYTOPENIA

(LOW WHITE-BLOOD CELL COUNT, LOW RED-BLOOD CELL COUNT, AND LOW PLATELET COUNT)



BASICS

OVERVIEW

- “Pan-” refers to “all” or “whole;” “cytopenia” is a decrease in number or lack of cells in the circulating blood
- Pancytopenia is the simultaneous development of a low white-blood cell count (known as “leukopenia”), low red-blood cell count, to which the bone marrow does not respond to produce more red-blood cells (known as “nonregenerative anemia”), and low platelet or thrombocyte count (known as “thrombocytopenia”)
- White-blood cells are the cells that protect the body from infection and disease; red-blood cells are the most numerous cells in blood—they carry oxygen to the tissues of the body; “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- Pancytopenia is not a disease itself, rather it is a laboratory finding that can result from multiple causes

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs related to underlying cause
- Repeated episodes of fever or frequent or persistent infections from the low white-blood cell count (leukopenia)
- Sluggishness (lethargy) or pale gums and moist tissues of the body (known as “pallor”) from the low red-blood cell count (anemia)
- Tiny, pinpoint bruises (known as “petechial hemorrhage”) or bleeding from the moist tissues of the body (known as “mucosal bleeding”) from the low platelet count (thrombocytopenia)
- Weakness
- Bleeding (for example, blood in the urine [known as “hematuria”]; bleeding from the nose [known as “epistaxis”]; spitting up of blood derived from the lungs due to pulmonary or bronchial hemorrhage [known as “hemoptysis”]; black, tarry stools due to the presence of digested blood [known as “melena”])
- Fever

CAUSES

Infectious Diseases

- Feline leukemia virus (FeLV)
- Feline immunodeficiency virus (FIV)
- Ehrlichiosis, a tick-borne disease
- Feline infectious peritonitis (FIP)
- Canine and feline parvovirus
- Infectious canine hepatitis virus
- Histoplasmosis, a fungal disease
- Accumulation of bacterial toxins in the blood (known as “endotoxemia”) and generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”), especially gram-negative organisms or tularemia (“rabbit fever”)

Drugs, Chemicals, and Toxins

- Estrogen (administration of estrogen-containing medications or secondary to tumors of the testicles [Sertoli cell tumor and interstitial cell tumor])
- Various medications, including phenylbutazone, griseofulvin, methimazole (cats), chloramphenicol, trimethoprim-sulfadiazine, albendazole, captopril, second-generation cephalosporins, and chemotherapeutic drugs (such as azathioprine, doxorubicin, carboplatin, cyclophosphamide, cytosine arabinoside, vinblastine, hydroxyurea)
- Poisons, such as thallium
- Radiation

Proliferative and Infiltrative Diseases

- Cancer of the blood and/or bone marrow (such as sudden (acute) and long-term (chronic) leukemias or lymphoma)
- Scar tissue build-up in the bone marrow (known as “myelofibrosis”)
- Replacement of the bone marrow by abnormal tissue, such as cancer (known as “myelophthisis”)
- Abnormal hardening of bone (known as “osteosclerosis”)

Immune-mediated Diseases

- Decreased ability of the bone marrow to produce red-blood cells (known as “aplastic anemia”) or to produce red-blood cells, white-blood cells, and platelets (known as “aplastic pancytopenia”)
- Immune-mediated hemolytic anemia and thrombocytopenia; “immune-mediated hemolytic anemia” is a low red-blood cell count due to the destruction of red-blood cells by the immune system and “immune-mediated thrombocytopenia” is a low platelet count due to the destruction of platelets by the immune system

RISK FACTORS

- Vary with individual underlying cause



TREATMENT

HEALTH CARE

- Supportive treatment depends on the clinical situation and includes aggressive antibiotic therapy and blood transfusions
- Treatment of the underlying condition is paramount



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treatment should be appropriate for the clinical situation (that is, the degree to which each type of blood cell is decreased, presence of fever or infection, and established or suspected specific diagnoses)
- Medications and treatment are directed at the underlying cause
- Medications to increase the blood-cell counts are “recombinant hematopoietic growth factors;” they include “granulocyte colony-stimulating factor” (filgrastim, Neupogen®) to stimulate the production of neutrophils (a type of white-blood cell that fights infection) and “erythropoietin” (epoetin, Epogen®) to stimulate the production of red-blood cells by the bone marrow



FOLLOW-UP CARE

PATIENT MONITORING

- Daily physical examination, including frequent monitoring of body temperature
- Periodic complete blood count (CBC)—frequency depends on severity of low white-blood cell count, red-blood cell count, and platelet count (cytopenia), age, general physical condition of the patient, and underlying cause

PREVENTIONS AND AVOIDANCE

- Castration of cryptorchid males; cryptorchid males have one or both testicles located in the abdomen or inguinal area, not in the scrotum
- Vaccination against infectious diseases
- Frequent monitoring of complete blood counts (CBCs) in cancer patients receiving chemotherapy

POSSIBLE COMPLICATIONS

- Bleeding
- Generalized bacterial infection (known as “sepsis”)

EXPECTED COURSE AND PROGNOSIS

- Depend on the underlying cause
- Often a guarded prognosis is warranted



KEY POINTS

- Pancytopenia is the simultaneous development of a low white-blood cell count (leukopenia), low red-blood cell count, to which the bone marrow does not respond to produce more red-blood cells (nonregenerative anemia), and low platelet or thrombocyte count (thrombocytopenia)
- Pancytopenia is not a disease itself, rather it is a laboratory finding that can result from multiple causes

PANOSTEITIS



BASICS

OVERVIEW

- A self-limiting, painful condition affecting one or more of the long bones of young, medium- to large-breed dogs that is characterized clinically by lameness and increased density of the marrow cavity on X-rays
- “Osteitis” is inflammation of bone; “panosteitis” is inflammation of an entire bone

GENETICS

- No proven transmission
- Predominance of German shepherd dogs in the affected population strongly suggests a genetic basis

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- German shepherd dogs and German shepherd mixes—most commonly affected
- Medium- to large-breed dogs—most commonly affected

Mean Age and Range

- Usually 5 to 18 months of age
- Affected dogs have been as young as 2 months and as old as 5 years of age

Predominant Sex

- Male

SIGNS/OBSERVED CHANGES in the ANIMAL

- Lameness—if no distinct abnormalities noted on physical examination or X-rays, repeat examination and X-rays 4 to 6 weeks later
- No history or signs of associated trauma
- Lameness—varying intensity; usually involves the front legs initially; may affect the hind legs; may see shifting leg lameness (that is, lameness in one leg and then in another leg); may be non-weight bearing
- Severe disease—mild depression; lack of appetite; weight loss
- Pain—on deep palpation of the shaft (known as the “diaphysis”) of the long bones in an affected limb; distinguishing characteristic; palpate firmly along the entire shaft of each bone while carefully avoiding any pinching of nearby muscle
- Bones—ulna in the lower front leg is affected most commonly; may affect radius (second bone in the lower front leg), humerus (bone in the upper front leg), femur (bone in the thigh), and tibia (bone in the lower rear leg)—in decreasing order of frequency—may affect bones either at the same time or subsequently
- May have low-grade fever
- May have loss of muscle mass (known as “muscle atrophy”)

CAUSES

- Unknown

RISK FACTORS

- Purebred German shepherd dog or German shepherd mixed-breed dog



TREATMENT

HEALTH CARE

- Outpatient

- Maintenance and replacement fluid therapy—occasionally needed, owing to prolonged periods of lack of appetite and fever (known as “pyrexia”)

ACTIVITY

- Limited—not shown to hasten recovery; lessens pain
- Moderate to severe disease—pain may cause self-limited movement, leading to muscle atrophy



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Symptomatic therapy has no bearing on the duration of the disease
- Nonsteroidal anti-inflammatory drugs (NSAIDs) minimize pain; decrease inflammation; include such drugs as carprofen, etodolac, meloxicam, deracoxib, firocoxib, buffered or enteric-coated aspirin—use only under the direction of your pet’s veterinarian
- Steroids to decrease inflammation, such as prednisone; goal for long-term use is low dose, alternate-day therapy



FOLLOW-UP CARE

PATIENT MONITORING

- Recheck lameness every 2 to 4 weeks
- Monitor for more serious orthopedic problems that may occur at the same time as panosteitis

EXPECTED COURSE AND PROGNOSIS

- Self-limiting disease
- Treatment—symptomatic; appears to have no influence on duration of clinical signs
- Multiple limb involvement—common
- Lameness—typically lasts from a few days to several weeks; may persist for months
- Occasional case has unrelenting pain and lameness that is unresponsive to therapy; euthanasia has been recommended in this situation



KEY POINTS

- Dog may develop other juvenile bone (orthopedic) diseases
- Signs of pain and lameness may last for several weeks
- Recurrence of clinical signs is common up to 2 years of age

PARALYSIS



BASICS

OVERVIEW

- Paresis—weakness of voluntary movement
- Paralysis—lack of voluntary movement
- Quadriparesis (tetraparesis)—weakness of voluntary movements in all legs
- Quadriplegia (tetraplegia)—absence of all voluntary movement in the legs
- Paraparesis—weakness of voluntary movements in the rear legs
- Paraplegia—absence of all voluntary rear leg movement
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae
- Degeneration of the intervertebral disks causes movement of disk material into the spinal canal; the disk material then causes spinal-cord compression (known as “myelopathy”) and/or nerve-root compression (known as “radiculopathy”)—so called “intervertebral disk disease”
- “Neurons” are nerve cells that join together to form nerves; “motor neurons” are nerve cells that control muscles
- Disease involving the nerve cells of the brain, brain stem and/or spinal cord that control the muscles is known as “upper motor neuron disease”
- Disease of the nerves that connect the spinal cord and muscles is known as “lower motor neuron disease”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Degenerative intervertebral disk disease—dachshunds, poodles, cocker spaniels, and beagles
- Sudden, rapidly progressive paralysis (known as “coonhound paralysis”)—hunting dogs
- Spinal cord and vertebral trauma—roaming animals
- Dislocation of the joint between the first and second cervical vertebra (condition known as “atlantoaxial luxation”)—toy and small breeds
- Pressure to or damage of the nerves within the spinal canal in the area of the junction between the lumbar and sacral vertebrae (known as “lumbosacral instability”); at this level of the spine, spinal nerves are located in the spinal canal (rather than spinal cord)—these spinal nerves within the spinal canal are known as the “cauda equina”—large breeds; working breeds; German shepherd dogs
- Condition in which the vertebrae in the neck are malformed, leading to narrowing of the spinal canal, or excessive mobility with resulting pressure on the spinal cord (known as “cervical vertebral malformation/stenosis/instability syndrome or wobbler syndrome”)—large breeds; Doberman pinschers; Great Danes
- Condition with abnormal cavities filled with fluid within the spinal cord (known as “syringomyelia”): Cavalier King Charles spaniels, Weimaraners
- Condition in which fluid-filled sacs are located under the arachnoid membrane (one of the protective membranes around the central nervous system [brain and spinal cord]) of the spinal cord (condition known as “spinal arachnoid cysts”): rottweilers, small breeds

SIGNS/OBSERVED CHANGES in the ANIMAL

- Limb weakness—sudden (acute) or gradual onset
- Being “down,” unable to move, walk, or get up (known as being “nonambulatory”)
- Signs may begin with a wobbly, incoordinated or “drunken” appearing gait (known as “ataxia”) and progress to weakness and finally to paralysis
- Usually alert
- If in pain, pet may resent handling and manipulation during physical examination
- Blood clots in the aorta, the main artery of the body (known as “aortic emboli”) leading to nervous tissue and muscle disease from lack

of blood flow (known as “ischemic neuromyopathy”)—patient may have paralysis of the rear legs (paraplegia) and lack of reflexes (known as “areflexia”) or decreased reflexes (known as “hyporeflexia”) on examination; femoral pulses absent; legs often cold; nail beds often blue

- If legs are paralyzed, likely that bladder is paralyzed as well; animal may not be able to urinate
- Location of problem in the spinal cord or nerves causing weakness or paralysis will determine signs observed in the pet and will aid in making a possible diagnosis

CAUSES

Generalized Paralysis of All Legs (Quadriplegia)

- Upper motor neuron (involves nerve cells of the brain, brain stem and/or spinal cord that control muscles)—cervical spinal cord disease or spinal cord disease involving multiple locations: intervertebral disk disease; bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition known as “diskospondylitis”); condition in which a piece of cartilage breaks off the intervertebral disk and travels in the blood vessel until it blocks blood flow to the spinal cord (known as “fibrocartilaginous embolism”); trauma; cancer; inflammation of the spinal cord (known as “myelitis”) of many causes; malformations of the spine or spinal cord
- Lower motor neuron (involves nerves that connect the spinal cord and muscles)—sudden (acute) onset: coonhound paralysis; botulism; tick paralysis (paralysis that develops due to the presence of a nerve toxin that enters the body through tick bites); severe, rapidly progressive form of myasthenia gravis (a disorder of neuromuscular transmission characterized by muscular weakness and excessive fatigue); or protozoal inflammation of the muscles and nerves (known as “protozoal myoneuritis”); more gradual onset: disorders characterized by inflammation of several nerves (known as “polyneuropathies”) and several muscles (known as “polymyopathies”) from toxicity, infection, inflammation, hormonal disease, metabolic disease, or congenital (present at birth)/inherited disease

Paralysis of the Rear Legs (Paraplegia)

- Upper motor neuron (involves nerve cells of the brain, brain stem and/or spinal cord that control muscles)—intervertebral disk disease; bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition is diskospondylitis); condition in which a piece of cartilage breaks off the intervertebral disk and travels in the blood vessel until it blocks blood flow to the spinal cord (fibrocartilaginous embolism); cancer; trauma; congenital (present at birth) malformations of spine or spinal cord; disease of the spinal cord that causes progressive weakness of the rear legs (known as “degenerative myelopathy”)
- Lower motor neuron (involves nerves that connect the spinal cord and muscles)—condition in which a piece of cartilage breaks off the intervertebral disk and travels in the blood vessel until it blocks blood flow to the spinal cord (fibrocartilaginous embolism); intervertebral disk disease; lumbosacral instability; bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition is diskospondylitis); trauma; cancer; defective development of the spine leading to exposure of the covering of the spinal cord (known as “meninges”) or spinal cord (condition known as “spina bifida”)

Generalized Paralysis of All Legs (Quadriplegia) with Cranial Nerve Deficits, Seizures, or Stupor

- Upper motor neuron (involves nerve cells of the brain, brain stem and/or spinal cord that control muscles)—diseases of the brain stem: inflammation of the brain (known as “encephalitis”); cancer; trauma; vascular accidents; congenital (present at birth) or inherited disorders

RISK FACTORS

- Breeds at risk for degenerative intervertebral disk disease—dachshunds, poodles, cocker spaniels, and beagles
- Hunting dogs—coonhound paralysis
- Roaming animals—spinal cord and vertebral trauma
- Toy and small breeds—atlandoaxial luxation
- Large breeds; working breeds; German shepherd dogs—lumbosacral instability
- Large breeds; Doberman pinschers; Great Danes—cervical vertebral malformation/stenosis/instability syndrome (wobbler syndrome)
- Cavalier King Charles Spaniels, Weimaraners—syringomyelia
- Rottweilers, small breeds—spinal arachnoid cysts



TREATMENT

HEALTH CARE

- Inpatient—with severe weakness or paralysis until bladder function can be determined
- Bladder—empty (via manual pressure or catheterization) three to four times a day to prevent overdistention and subsequent complications; once bladder function has returned, patient can be managed at home
- Bedding—move paralyzed patients away from soiled bedding; check and clean frequently to prevent skin lesions that develop due to contact with urine, when the hair and skin remain damp (known as “urine scald”) and superficial skin infection characterized by the presence of pus (known as “superficial pyoderma”); use padded bedding or a waterbed to help prevent “bed sores” (known as “decubital ulcers”)

- Turning—turn patients that are paralyzed in all legs (quadriplegia) from side to side four to eight times daily; prevent lung congestion and “bed sore” (decubital ulcer) formation

ACTIVITY

- Activity—restrict until spinal trauma and intervertebral disk disease can be ruled out
- Physical therapy—important for paralyzed patients; tones muscles and keeps joints flexible

DIET

- Hand feeding—with widespread (diffuse) lower motor neuron (involves nerves that connect the spinal cord and muscles) signs, swallowing can be affected; carefully hand feed until it is certain that the patient can swallow properly
- Feeding from an elevated platform or installation of a feeding tube—recommended for animals with enlargement of the esophagus (the tube running from the mouth to the stomach; condition known as “megaesophagus”) until it resolves

SURGERY

- Surgery—for intervertebral disk disease, fracture, some tumors and some congenital (present at birth) conditions; often the quickest and most effective method of improving the nervous system status



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroid use (even in known diseases like spinal trauma or intervertebral disk disease) is somewhat controversial; current information suggests that steroids may be helpful to decrease pain associated with some spinal cord causes of paralysis, but that they do not enhance spinal cord recovery; include dexamethasone, prednisolone
- Pyridostigmine bromide—for suspected myasthenia gravis (a disorder of neuromuscular transmission characterized by muscular weakness and excessive fatigue)
- Sudden (acute) generalized lower motor neuron (involves nerves that connect the spinal cord and muscles) signs—check for ticks; treat with appropriate insecticides, if necessary
- Nonsteroidal anti-inflammatory drugs (NSAIDs) for spinal diseases that might be associated with bone discomfort or pain
- Tramadol for pain relief
- Gabapentin for pain arising from a disorder of the nervous system (known as “neuropathic pain”)
- Butorphanol for pain control



FOLLOW-UP CARE

PATIENT MONITORING

- Nervous system examinations—daily to monitor status

PREVENTIONS AND AVOIDANCE

- Prevent tick infestation (control ticks in the environment and on the animal)
- Keep the pet in a safe environment (in the house, in a fenced yard, on a leash) to prevent accidents (such as being hit by a car) that may lead to spinal fractures

POSSIBLE COMPLICATIONS

- Urinary tract infection
- Flaccid bladder (known as “bladder atony”), in which the bladder muscles do not contract normally
- Skin lesions that develop due to contact with urine, when the hair and skin remain damp (urine scald) and skin infection characterized by the presence of pus (pyoderma)
- Constipation
- “Bed sores” (decubital ulcers)
- Aspiration pneumonia—with generalized lower motor neuron disease (involves nerves that connect the spinal cord and muscles) or in any patient that is paralyzed in all four legs (quadriplegia)

- Condition in which the motor neurons (nerve cells that control muscles) are destroyed, leading to progressive spinal cord disease that is not reversible (condition known as “myelomalacia”)—with severe spinal cord trauma or intervertebral disk disease
- Breathing compromise or paralysis—with destruction of the motor neurons (nerve cells that control muscles) and progressive spinal cord disease (myelomalacia) or generalized lower motor neuron disease (involves nerves that connect the spinal cord and muscles)

EXPECTED COURSE AND PROGNOSIS

- Depend on cause of weakness or paralysis



KEY POINTS

- Empty (via manual pressure or catheterization) the bladder of paralyzed pets three to four times a day to prevent overdistention and subsequent complications; once bladder function has returned, patient can be managed at home
- Bedding—move paralyzed pets away from soiled bedding; check and clean frequently to prevent skin lesions that develop due to contact with urine, when the hair and skin remain damp (urine scald) and superficial skin infection characterized by the presence of pus (superficial pyoderma); use padded bedding or a waterbed to help prevent “bed sores” (decubital ulcers)
- Turning—turn patients that are paralyzed in all legs (quadriplegia) from side to side four to eight times daily; prevent lung congestion and “bed sore” (decubital ulcer) formation

RESPIRATORY PARASITES



BASICS

OVERVIEW

- Parasites are organisms that live, grow, and feed on or in another organism; “helminths” are worms, such as roundworms or tapeworms; “arthropods” are animals without backbones (invertebrates) that have a hard, external skeleton (exoskeleton), such as insects
- Respiratory parasites are helminths and arthropods that reside in the respiratory tract or in the blood vessels of the lungs (known as “pulmonary vessels”) of dogs and cats
- “Upper respiratory tract” consists of the nose, nasal passages, throat, and windpipe (trachea)
- “Lower respiratory tract” consists of the bronchi, bronchioles, and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Three basic categories of signs—upper respiratory signs that involve the nose, nasal passages, throat, and windpipe (trachea); lower respiratory signs that involve the bronchi, bronchioles, and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged); and vascular signs that involve the blood vessels to the lungs; signs are based on location and lifestyle of parasite
- Often subtle and long-term (chronic) disease, with few clinical signs
- Breathing problems often not severe
- Upper respiratory signs—sneezing; watery or bloody discharge from the nose; reverse sneezing; nasal irritation or rubbing the nose; nervous system signs with *Cuterebra* (*Cuterebra* is a fly; it lays eggs near rodent burrows and when an animal moves in the area, the eggs hatch releasing larvae that enter the animal; the larvae develop under the skin or in the nose and mouth; they occasionally migrate through the brain)
- Lower respiratory signs—cough that can be stimulated by handling or pressure on windpipe (trachea); occasionally harsh lung sounds; often cause coughing in cats

CAUSES

- Upper respiratory disease—variety of parasites, including *Pneumonyssoides caninum* (nasal mites); *Eucoleus boehmi* (nasal worm); *Linguatula serrata*
- Lower respiratory disease—variety of parasites in dogs and cats, including *Capillaria aerophila* (rare in cats), *Paragonimus kellicotti* (lung fluke), *Eucoleus aerophilus*; and parasites in dogs: *Oslerus osleri* (*Filaroides osleri*), *Filaroides hirthei*, *Filaroides milksi*, *Crenosoma vulpis*; as well as parasites in cats: *Aelurostrongylus abstrusus*, *Cuterebra*, larval migration of *Toxocara canis*

RISK FACTORS

- Depends on the specific parasite—some have intermediate hosts (animal hosts [such as mosquitoes for heartworms] in which the parasite must undergo part of its life cycle, prior to infecting the dog or cat) or paratenic hosts (animal hosts in which the parasite may grow or develop, but this host is not required in the life cycle of the parasite; the dog or cat becomes infected after eating the paratenic host; for example, dogs and cats can become infected with *Paragonimus kellicotti* (lung fluke) after eating a crayfish carrying the immature form of the lung fluke); exposure to these hosts increases the risk of infestation with the parasite, putting hunting or scavenging animals at higher risk
- *Crenosoma vulpis*—snails, paratenic host
- *Paragonimus kellicotti*—snails; crabs; shellfish
- *Aelurostrongylus abstrusus*—snails and slugs; transport hosts: rodents, frogs, lizards, birds
- *Linguatula serrata*—ingestion of sheep offal (waste parts of a butchered animal)
- Multi-animal households with unhealthy living conditions—allows transmission of parasites through eating feces-contaminated materials or feces (fecal–oral transmission) or through direct contact with the infective stage of the parasite (direct-contact transmission)



TREATMENT

HEALTH CARE

- Outpatient—upper and lower respiratory parasites; may need repeated examinations to monitor response

ACTIVITY

- No restrictions, unless severe lung/breathing difficulties occur with upper or lower respiratory parasites

DIET

- No special restrictions



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drugs to kill worms (known as “anthelmintics”)—few studies confirm effectiveness for respiratory parasites
- *Pneumonyssoides caninum*—ivermectin (suggested dosage for treatment of respiratory parasites contraindicated in collies, collie breeds, and Australian shepherds because of high incidence of toxicity); milbemycin oxime; selamectin
- *Aelurostrongylus abstrusus*—fenbendazole; possibly selamectin
- Other upper and lower respiratory parasites—fenbendazole; variable success with ivermectin
- *Crenosoma vulpis*—levamisole; fenbendazole; febantel
- *Paragonimus kellicotti*—praziquantel; albendazole
- *Cuterebra*—ivermectin
- Anti-inflammatory agents—generally not required; may reduce effectiveness of drugs used to kill the worms (anthelmintics)



FOLLOW-UP CARE

PATIENT MONITORING

- Repeated examination for parasite eggs or larvae—some drugs used to kill the worms (anthelmintics) may suppress egg or larval production in some species of parasite
- Resolution of clinical signs—suggests response to treatment; does not indicate complete clearance of parasites
- Presence of high eosinophil (a type of white-blood cell) counts in the circulating blood (known as “peripheral eosinophilia”), if noted initially, may subside with treatment
- Repeat examination of the airways using a special lighted instrument called an “bronchoscope”—may help assess effectiveness of treatment for *Oslerus osleri*

PREVENTIONS AND AVOIDANCE

- Avoid activity that increases likelihood of parasite infestations (often not practical)
- Avoid contact with wildlife reservoirs (especially wild canids and felids)
- Consider prophylactic treatment for heartworm
- *Pneumonyssoides caninum* infestation appears to be lower in animals taking heartworm preventive medication, suggesting that ivermectin prevents infestation

POSSIBLE COMPLICATIONS

- Long-term (chronic) lung (pulmonary) damage—possible with persistent and heavy lower respiratory parasite burdens
- Infestations generally not fatal; however, severe lung damage can result with some species; *Cuterebra* can cause fatal nervous system complications

- *Pneumonyssoides caninum* has been associated with a condition in which the stomach dilates with gas and/or fluid (known as “gastric dilatation”), and subsequently rotates around its short axis (known as “volvulus”)—condition known as “gastric dilatation-volvulus” or “bloat”)

EXPECTED COURSE AND PROGNOSIS

- Prognosis is variable; with aggressive management—prognosis usually fair to excellent
- Return to performance—depends on long-term duration of disease and level of long-term (chronic) lung (pulmonary) damage by lower respiratory parasites
- Recurrence possible



KEY POINTS

- Treatment response and duration depend on the type of parasite
- Risk of recurrence in dogs that maintain lifestyles conducive to transmission of the parasites (such as hunting, sporting dogs, and multi-dog households, out-door cats)

CANINE PARVOVIRUS INFECTION



BASICS

OVERVIEW

- Canine parvovirus (CPV) infection is characterized clinically by lack of appetite, vomiting, diarrhea, and weight loss; severe disease may result in generalized bacterial infection (known as “sepsis”), presence of bacterial toxins in the blood (known as “endotoxemia”), blood clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”), and acute respiratory distress syndrome (ARDS)
- The original canine parvovirus underwent genetic alterations, developing into CPV-1 and CPV-2; CPV-2 developed further into CPV-2a in 1980 and CPV-2b in 1984
- Most severe disease is associated with CPV-2b
- CPV-1 may cause unmanageable, usually fatal diarrhea in newborn puppies
- Recently CPV-2b has shown mutation into a variant form designated Glu426

GENETICS

- Unknown

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Cats—can be infected with CPV-2b

Breed Predilections

- Rottweilers, Doberman pinschers, pit bulls, Labrador retrievers, German shepherd dogs, English springer spaniels, Alaskan sled dogs are considered to be more susceptible to canine parvovirus infection than are other breeds of dog

Mean Age and Range

- Most cases are seen between 6 weeks and 6 months of age
- More severe disease is seen in younger puppies
- Incidence has decreased dramatically with vaccination of puppies against parvovirus

SIGNS/OBSERVED CHANGES in the ANIMAL

- Loss of energy, sluggishness (lethargy), lack of appetite (anorexia), vomiting, and profuse diarrhea with rapid, severe weight loss
- Rapid heart rate (tachycardia)
- Moist tissues of mouth and eyes (mucous membranes) may be pale or deep red, due to the blood vessels being filled with blood (injected), or yellowish (icteric)
- Dehydration
- Pain or discomfort when the veterinarian feels the abdomen (known as “abdominal palpation”)
- Intestines may be fluid filled, or rarely, the veterinarian may detect the folding of one segment of the intestine into another segment (known as “intussusception”)
- May have a fever or the body temperature may be lower than normal (hypothermia)
- May exhibit vomiting/diarrhea in the examination room

CAUSES

- CPV-2b (Canine Parvovirus-2b) infection

RISK FACTORS

- Breed predisposition as listed under “Breed Predilection”
- Possible simultaneous conditions, diseases or drug therapy that lead to an inability to develop a normal immune response (known as “immunosuppression”), such as heavy parasitism
- Incomplete vaccination protocol, vaccine failure, or normal interference of the puppy developing protective antibodies due to the presence of maternal antibodies
- Breeding kennels, pounds, shelters, and areas with a high number of puppies without adequate immune response or inadequately vaccinated puppies



TREATMENT

HEALTH CARE

- Hospitalization for intensive therapy and supportive treatment significantly improves survival
- Hospitalized cases must be kept isolated from other patients; hospital personnel must follow proper cleaning and disinfecting practices

to prevent spread of the virus

- Intravenous fluid therapy is a mainstay of treatment; fluid rates must account for maintenance needs plus ongoing losses, which may be profound due to vomiting and diarrhea
- Colloid therapy (using certain fluids with larger molecular weight substances than found in typical IV fluids to expand the plasma volume) may be necessary in patients with low levels of albumin (a protein) in the blood (condition known as “hypoalbuminemia”)
- Transfusions with plasma or hyperimmune serum may be used

ACTIVITY

- Activity should be restricted until puppies are recovering

DIET

- Food and water should be withheld if vomiting
- Small amounts of water may be introduced after 24 hours with no vomiting
- Nutrition utilizing some type of feeding tube (known as “enteral or microenteral nutrition”) should be considered in cases with lack of appetite (anorexia) of 3 to 4 days’ duration; early enteral nutrition may improve clinical outcome
- Providing nutrition via intravenous therapy (known as “parenteral nutrition”) may be required in severe cases
- Glutamine supplementation has been shown to improve the health of intestinal cells
- A bland, easily digestible diet (such as Hill’s Prescription Diet® i/d®, Purina Veterinary Diets® EN®) should be fed initially, with gradual transition to the normal ration

SURGERY

- The only surgical indication is for treatment of the rare complication of intestinal intussusception (the folding of one segment of the intestine into another segment)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drugs to stop vomiting (antiemetics)—very frequently needed due to prolonged vomiting; examples include metoclopramide; phenothiazines, such as promethazine; serotonin receptor antagonists, such as ondansetron
- H₂-blockers—may reduce nausea; such as cimetidine; ranitidine; famotidine
- Antibiotics—to combat generalized bacterial infection (sepsis); should have spectrum to include gram-negative organisms
- Medications (known as “anthelmintics”) to eradicate intestinal parasites
- Pain relievers (analgesics)—may be needed in severe cases
- Anecdotal reports describe the use of equine endotoxin antiserum; no controlled studies demonstrate a survival benefit with this therapy
- Recent studies have shown no survival benefit in using granulocyte colony-stimulating factor, anti-tumor necrosis factor (antiTNF), or recombinant bactericidal/permeability-increasing protein (rBPI₂₁)
- Activated protein C and interferon (IFN) Ω may be promising future treatment options
- Less severely affected puppies may be managed on an outpatient basis with fluids administered under the skin (subcutaneous) and/or into the abdominal cavity (intraperitoneal), if owner has financial constraints



FOLLOW-UP CARE

PATIENT MONITORING

- Aggressive therapy improves survival, but mortality rates may still approach 30%.
- If the puppy recovers, recovery is typically complete; immunity following canine parvovirus infection is long term and may be lifelong

PREVENTIONS AND AVOIDANCE

- Vaccination against canine parvovirus has been effective at drastically reducing disease incidence
- Modified live (high-titer) vaccines are recommended for puppies to minimize interference from maternal antibodies
- Interference from maternal antibodies is the main reason for vaccine failure; some puppies may have maternal antibodies present in their blood for up to 18 weeks of age
- Protocols recommend vaccinating at 6, 9, and 12 weeks of age
- High-risk breeds may require a longer initial vaccination protocol against canine parvovirus, extending up to 22 weeks of age
- Recent studies indicate immunity may last 3+ years after completion of the initial vaccine protocol

POSSIBLE COMPLICATIONS

- Generalized bacterial infection (sepsis)

- Presence of bacterial toxins in the blood (endotoxemia)
- Shock
- Intussusception (the folding of one segment of the intestine into another segment)
- Blood clotting disorder (disseminated intravascular coagulopathy or DIC)
- Acute respiratory distress syndrome (ARDS)

EXPECTED COURSE AND PROGNOSIS

- Mortality is primarily due to the presence of bacterial toxins in the blood (endotoxemia)
- Aggressive therapy improves survival, but mortality rates may still approach 30%



KEY POINTS

- Canine parvovirus is very stable in the environment, but may be destroyed by use of 1:30 bleach solution
- Vaccine does not produce immediate immunity, so susceptible puppies should be kept isolated
- Mortality is primarily due to the presence of bacterial toxins in the blood (endotoxemia)
- Aggressive therapy improves survival, but mortality rates may still approach 30%

PATELLAR LUXATION

(KNEECAP DISLOCATION)



BASICS

OVERVIEW

- The “patella” is the kneecap; it is located at the front of the stifle joint; the “stifle” is the knee joint of the dog or cat—it is the joint between the large upper thigh bone (the femur) and the two lower leg bones (tibia and fibula)
- “Luxation” is the medical term for dislocation
- “Patellar luxation” is the displacement of the patella from its normal anatomic position in the groove of the femur (known as the “femoral trochlea”); the displacement can be to the inner side of the stifle (known as a “medial patellar luxation”) or to the outer side of the stifle (known as a “lateral patellar luxation”)
- One of the most common stifle-joint abnormalities in dogs
- Medial patellar luxation (that is, dislocation toward the inner side of the stifle)—greater than 75% of cases involving large and small dogs and cats
- Involvement of both kneecaps (known as “bilateral patellar luxations”)—seen in 50% of cases
- Uncommon in cats; however, may be more common than suspected because most affected cats are not lame

GENETICS

- Recessive, multiple genes (known as a “polygenic trait”), and multifocal inheritances proposed
- Hereditary factor in Devon rex cats

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Predominantly dogs
- Uncommon in cats

Breed Predispositions

- Most common in toy and miniature dog breeds
- Dogs—miniature and toy poodles; Yorkshire terriers; Pomeranians; Pekingese; Chihuahuas; Boston terriers

Mean Age and Range

- Clinical signs—may develop soon after birth; generally after 4 months of age

Predominant Sex

- Risk for females is 1.5 times that for males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical expression depends on grade (severity), amount of degenerative joint disease (progressive and permanent deterioration of joint cartilage), long-term nature (chronicity) of disease, and occurrence of other stifle joint abnormalities (such as cranial cruciate ligament rupture [partial or complete tearing of the cranial cruciate ligament])
- Persistent abnormal rear-leg carriage and function in newborns and puppies
- Occasional skipping or intermittent rear-leg lameness—worsens in young to mature dogs
- Sudden signs of lameness—owing to minor trauma or worsening degenerative joint disease (progressive and permanent deterioration of joint cartilage) in mature animals
- Pain—occurs as the kneecap (patella) moves in the abnormal position or if it contacts or rubs exposed bone

Grades of Patellar Luxation

- Grade I—kneecap (patella) can be displaced manually from its normal location; but immediately resumes a normal position when pressure is released
- Grade II—kneecap (patella) can be displaced manually or can displace spontaneously with bending (flexion) of the stifle joint; patella remains in its displaced location until it is replaced manually or the pet straightens (extends) the stifle joint; pet intermittently carries the affected leg with the knee (stifle) joint flexed
- Grade III—kneecap (patella) remains dislocated most of the time, but can be replaced manually when the stifle joint is straightened (extended); movement of the stifle joint results in re-dislocation of the patella
- Grade IV—kneecap (patella) is dislocated permanently and cannot be replaced manually
- Grades III and IV—crouching, bowlegged or knock-kneed stance for medial (that is, dislocation toward the inner side of the stifle) or

lateral (that is, dislocation toward the outer side of the stifle) luxations, respectively; most of the body weight is transferred to the front limbs

CAUSES

- Congenital (present at birth)
- Trauma



TREATMENT

HEALTH CARE

- Outpatient—all grade I and some grade II kneecap (patella) dislocations (patellar luxations)
- Inpatient for surgery—most grade II and all grade III and IV kneecap (patella) dislocations (patellar luxations)
- Ice packing (known as “cryotherapy”)—immediately after surgery; apply ice packs for 5 to 10 minutes every 8 hours for 3 to 5 days or as directed by your pet’s veterinarian
- Passive stifle range-of-motion exercises—as soon as tolerated

ACTIVITY

- Normal to restricted, depending on severity of kneecap (patella) dislocation (patellar luxation)
- Following surgery—encourage early, active use of the limb; leash walk exercise for 4 weeks; prevent jumping

DIET

- Weight control—decreases stress on the kneecap (patella)-support mechanism

SURGERY

- Various surgical procedures may be performed; type of surgery determined by anatomy of the stifle and the severity of the kneecap (patella) dislocation (patellar luxation)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs)—minimize pain; decrease inflammation; examples include meloxicam, carprofen, etodolac, deracoxib
- Medications intended to slow the progression of arthritic changes and protect joint cartilage (known as “chondroprotective drugs”), such as polysulfated glycosaminoglycans, glucosamine, and chondroitin sulfate—may help limit cartilage damage and degeneration; may help alleviate pain and inflammation



FOLLOW-UP CARE

PATIENT MONITORING

- Yearly examinations

PREVENTIONS AND AVOIDANCE

- Discourage breeding of affected animals
- Do not repeat dam–sire breedings that result in affected offspring

POSSIBLE COMPLICATIONS

- Recurrence of kneecap (patella) dislocation (patellar luxation) after surgical stabilization—reported to be as high as 48%; usually of a lower grade than the original patellar luxation

EXPECTED COURSE AND PROGNOSIS

- With surgical treatment—greater than 90% of patients are free from lameness and clinical dysfunction
- Degenerative joint disease (progressive and permanent deterioration of joint cartilage)—X-ray evidence in almost all affected stifle joints after surgery; however, clinical impact appears minimal in small dogs



KEY POINTS

- Congenital (present at birth) kneecap (patella) dislocation (patellar luxation) may have a genetic basis
- Potential exists for relapse following surgery; recurrence of patellar luxation after surgical stabilization reported to be as high as 48%
- Pets with patellar luxation may be at increased risk of cranial cruciate ligament disease (failure of the cranial cruciate ligament, which results in partial to complete instability of the stifle joint)
- Patellar luxation could worsen over time (such as from grade I to grade II)

PATENT DUCTUS ARTERIOSUS

(TYPE OF HEART BIRTH DEFECT)



BASICS

OVERVIEW

- “Patent” refers to “open;” “ductus arteriosus” is a blood vessel between the aorta (main artery of the body) and the pulmonary artery (main artery to the lungs) that allows blood flow to bypass the lungs in the fetus
- Following birth, the ductus arteriosus closes and seals off so that blood flows into the lungs, where it gets oxygen and allows carbon dioxide to be removed from the body
- “Patent ductus arteriosus” occurs when the open blood vessel persists and does not close following birth; this allows continued blood flow between the aorta and the pulmonary artery, which leads to abnormal circulation of blood in the body
- Patent ductus arteriosus also known as “PDA”
- Flow across a PDA is typically from the aorta to pulmonary artery (that is left to right flow)
- Much less frequently, a large-diameter PDA causes changes in the lungs (including high blood pressure in the lungs [known as “pulmonary hypertension”]), and reversal of blood flow occurs, leading to right to left flow; known as a “reversed” PDA
- Patent ductus arteriosus is the second most common congenital (present at birth) heart defect in dogs; estimated to be up to 2.5 cases per 1,000 live births
- Patent ductus arteriosus is a very uncommon heart defect in cats
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles

GENETICS

- Genetically transmitted defect involving multiple genes (known as a “polygenic” condition) in many canine breeds, including the Chihuahua, collie, miniature poodle, Maltese, English springer spaniel, bichon frise, Shetland sheepdog, German shepherd dog, cocker spaniel, Pomeranian, Cavalier King Charles spaniel, and Labrador retriever

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Many canine breeds, including the Chihuahua, collie, miniature poodle, Maltese, English springer spaniel, bichon frise, Shetland sheepdog, German shepherd dog, cocker spaniel, Pomeranian, Cavalier King Charles spaniel, and Labrador retriever

Mean Age and Range

- Vast majority of cases identified during examination for initial vaccinations
- Onset of signs related to congestive heart failure (signs such as cough, difficulty breathing, bluish discoloration of the skin and moist tissues [mucous membranes] of the body); “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs—weeks to many years of age

Predominant Sex

- Dogs—females more likely to have patent ductus arteriosus than males in many breeds

SIGNS/OBSERVED CHANGES in the ANIMAL

- Breathing distress
- Coughing
- Stunted growth
- Exercise intolerance; signs usually precipitated by or worsened by exercise
- Typically, continuous, machinery-type heart murmur loudest over pulmonary artery at the left base of the heart; the murmur in cats or in puppies less than 6 weeks of age may not be obviously continuous
- Loud murmurs—may be able to feel vibrations caused by abnormal blood flow (known as “thrills”) when placing hand against the chest wall
- Jerky arterial pulses (known as “water hammer” pulses)
- Rapid breathing (known as “tachypnea”), breathing distress, and inspiratory short, rough snapping sounds (known as “crackles”) heard when listening to the chest with a stethoscope—may indicate left-sided congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

- Rapid, irregular heart beat, if atrial fibrillation develops; “atrial fibrillation” is a rapid, irregular heart rhythm involving the top two chambers of the heart (atria)
- Onset of “reversed” PDA (in which blood flows from right to left)—quite sudden in dogs (usually before 4 months of age); can develop more gradually in cats
- In right-to-left shunting or “reversed” PDA—no continuous heart murmur is present and arterial pulses are normal; may have a heart murmur or abnormal heart sounds; may see a jugular pulse (in which the jugular vein pulsates)
- Classic feature of right-to-left shunting or “reversed” PDA is differential bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); with “reversed” PDA, the tissues near the head usually remain pink, while tissues toward the rear of the body have bluish discoloration (cyanosis)
- Right-to-left shunting or “reversed” PDA—exertional rear limb weakness and complications of increased red-blood cell counts (known as “polycythemia”) and sludging of the blood (known as “hyperviscosity”), seizures, sudden death related to irregular heart beats (known as “arrhythmias”), and blood clots that lodge in blood vessels (known as “emboli”)

CAUSES

- Genetic in most cases

RISK FACTORS

- Genetics in dogs
- Risk factors in cats are unknown



TREATMENT

HEALTH CARE

- Manage fluid build-up in the lungs (known as “pulmonary edema”) with medications to remove excess fluid from the body (known as “diuretics,” such as furosemide) and, if necessary, oxygen, medications to dilate blood vessels (known as “vasodilators,” such as nitrates), and cage rest
- Following stabilization, surgically correct the PDA promptly
- Can schedule stable animals for elective surgery or device closure; do not delay procedure—asymptomatic dogs as young as 7 to 8 weeks of age show no higher surgical mortality rates than older dogs
- Consider referral to a veterinary heart specialist (known as a “veterinary cardiologist”) for placement of a catheter-delivered device within the opening of the blood vessel to block blood flow through the PDA
- Application of an electrical shock to the chest (known as “electrical cardioversion”) to attempt to return the heart to normal rhythm may be considered in dogs with atrial fibrillation (rapid, irregular heart rhythm involving the top two chambers of the heart [atria]) following surgical correction of the PDA
- Dogs with increased red-blood cell counts (polycythemia) caused by right-to-left shunting or “reversed” PDA—periodic phlebotomy (procedure in which blood is removed from the body via a vein) to maintain the packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood) less than 65% (typically 62% to 65%)

ACTIVITY

- Depends on your pet’s condition

DIET

- Normal usually
- Restricted sodium intake, if pet is in congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

SURGERY

- Surgical closure of a PDA can be achieved through tying off the blood vessel after opening the chest (surgical opening of the chest known as a “thoracotomy”) to reach the location of the PDA or using a special lighted instrument called an “endoscope” (general term for procedure is “endoscopy”) to visualize the PDA
- Another technique is to place a catheter-delivered device within the opening of the blood vessel; the device is left in place to block blood flow through the ductus arteriosus; smaller patient size may be a limiting factor with ability to use currently available devices
- Surgery generally can proceed within 24 to 48 hours of medical stabilization
- A right-to-left or “reversed” PDA should not be corrected surgically; the right ventricle will not be able to pump blood against the pressure within the blood vessels of the lungs, without the “pop-off valve” effect of the PDA



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treat fluid build-up in the lungs (pulmonary edema) with medications to remove excess fluid (medications known as “diuretics,” such as furosemide); diuretics can be discontinued when the PDA is corrected surgically
- Pain management is appropriate and also shortens the post-operative recovery time
- When surgery is not an option—use medications to remove excess fluid (such as furosemide) and heart medications (such as enalapril and digoxin or pimobendan) to control congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- To control severe, life-threatening congestive heart failure—can use medications to enlarge or dilate blood vessels (vasodilators), such as hydralazine or sodium nitroprusside
- Prostaglandin inhibitors (such as indomethacin) do *not* close PDAs effectively in dogs; prostaglandin inhibitors have been used in treatment of PDA in human infants—prostaglandins are substances that have numerous effects on body function; a prostaglandin is involved in keeping the ductus arteriosus open during human fetal development, and the levels of the prostaglandin normally decrease after birth, allowing the blood vessel to close
- Consider hydroxyurea to treat severely increased red-blood cell counts (polycythemia) unresponsive to phlebotomy (procedure in which blood is removed from the body via a vein)



FOLLOW-UP CARE

PATIENT MONITORING

- Postoperative—monitor vital signs and difficulty breathing (dyspnea), which may be related to the presence of air in the space between the lungs and chest wall (condition known as “pneumothorax”)
- Listen to the heart with a stethoscope (known as “cardiac auscultation”) postoperatively and at suture removal; if sounds are normal, no further follow-up or diagnostic studies may be required
- Persistent, continuous murmur indicates either incomplete closure of the ductus arteriosus, reopening of the PDA (rule out infection or device migration), or a coexistent heart defect
- Systolic heart murmurs variably heard postoperatively should resolve by time of suture removal; reinvestigate unexpected heart murmurs by performing a Doppler echocardiogram (use of ultrasound to evaluate the heart and major blood vessels)
- Sudden (acute) illness, fever, or breathing difficulties (dyspnea) postoperatively—consider bacterial infection of the closure site with pneumonia; aggressive antibiotic therapy is needed

PREVENTIONS AND AVOIDANCE

- Do not breed affected animals

POSSIBLE COMPLICATIONS

- Left-sided congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Irregular heart beats (arrhythmias)
- Blood-vessel disease in the lungs with high blood pressure (pulmonary hypertension), right-to-left or “reversed” blood flow through the PDA, exercise intolerance, and increased red-blood cell counts (polycythemia)
- Reopening of the ductus arteriosus, following corrective procedures (surgery or catheter-delivered device)
- Problems with the catheter-delivered device (such as the device moving from its desired location or infection)
- Death

EXPECTED COURSE AND PROGNOSIS

- Infrequently dogs may not have any clinical signs during their life
- Unless the PDA is corrected (surgery or catheter-delivered device), approximately 50% to 60% of dogs die from congestive heart failure within 1 year of diagnosis
- Surgery performed prior to onset of moderate-to-severe congestive heart failure—excellent prognosis overall; approximately 5% of

cases die during surgery or in the post-operative recovery period in hospitals with veterinarians experienced in performing the corrective procedures

- Moderate-to-severe congestive heart failure related to failure of the muscle of the left ventricle or atrial fibrillation (rapid, irregular heart rhythm involving the top two chambers of the heart [atria])—guarded prognosis; consider referral to a veterinary heart specialist (veterinary cardiologist)
- Dogs with right-to-left or “reversed” PDA can live for several years, but often die suddenly; infrequently, dogs live beyond 5 years of age (especially cocker spaniels)
- Cats—varies from rapidly progressive left-sided congestive heart failure to gradual development of blood-vessel disease in the lungs; even right-sided congestive heart failure can develop in some of these cats



KEY POINTS

- Do not delay corrective procedures (surgery or catheter-delivered device) for treatment of PDA
- Following successful correction and a 2-week convalescence, the dog can be treated normally

PEMPHIGUS



BASICS

OVERVIEW

- A group of diseases in which the immune-system attacks the skin (known as “autoimmune dermatoses”); autoimmune diseases are ones in which the body produces antibodies against its own tissue; an “antibody” is a protein that is produced by the immune system in response to a specific antigen (a substance that induces an immune response)—when the body is exposed to the antigen (in the case of pemphigus, the antigen is some part of the skin), the antibody responds, resulting in signs of disease
- The pemphigus group of diseases is characterized by varying degrees of loss of tissue on the surface of the skin, frequently with inflammation (known as “ulceration”); dried discharge on the surface of a skin lesion (known as a “crust”); and formation of small, raised skin lesions containing pus (known as “pustules”) and blisters or small, circumscribed elevation of the outer layer of the skin filled with clear fluid (known as “vesicles”)
- Affects the skin and sometimes the moist tissues of the body (known as “mucous membranes”)
- Diseases include pemphigus foliaceus, pemphigus erythematosus, pemphigus vulgaris, and pemphigus vegetans; type of pemphigus based on location of skin lesions and microscopic appearance of skin lesions

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Pemphigus foliaceus, erythematosus, and vulgaris—dogs and cats
- Pemphigus vegetans—dogs only

Breed Predilections

- Pemphigus foliaceus—Akitas, bearded collies, chow chows, dachshunds, Doberman pinschers, Finnish spitzes, Newfoundlands, and schipperkes
- Pemphigus erythematosus—collies, German shepherd dogs, and Shetland sheepdogs

Mean Age and Range

- Usually middle-aged to old animals

SIGNS/OBSERVED CHANGES in the ANIMAL

Pemphigus Foliaceus

- Scales (accumulations of surface skin cells, such as seen in dandruff); crusts (dried discharge on the surface of skin lesions); pustules (raised skin lesions containing pus); superficial loss of skin tissue (known as “erosions”); reddened skin (known as “erythema”); hair loss (known as “alopecia”); circular patterns of hair loss (alopecia) bordered by scales or surface peeling of the skin (pattern is known as an “epidermal collarette”); and thickening of the skin (known as “hyperkeratosis”) of the footpads with furrows or slits (known as “fissures”)
- Occasional blisters (vesicles) are transient
- Common involvement—head, ears, and footpads; often becomes generalized
- Lesions involving the moist tissues of the body (mucous membranes) and areas where the moist tissues of the body contact the skin, such as the lips (areas known as “mucocutaneous junctions”) are uncommon
- Cats—nipple and nail-bed involvement are common
- Sometimes enlarged lymph nodes (known as “lymphadenopathy”), fluid build-up in the skin (known as “edema”), depression, fever, and lameness (if footpads involved) may be present; however, patients are often in good health
- Variable pain and itchiness (known as “pruritus”)
- Secondary bacterial infection is possible

Pemphigus Erythematosus

- Same signs as for pemphigus foliaceus
- Lesions usually confined to head, face, and footpads
- Loss of pigment of the moist tissues (mucous membranes) and skin (known as “mucocutaneous depigmentation”) more common than with other forms of pemphigus

Pemphigus Vulgaris

- Ulcerative lesions; superficial loss of skin tissue (erosions); circular patterns of hair loss (alopecia) bordered by scales or surface peeling of the skin (pattern is called epidermal collarettes), blisters, and crusts (dried discharge on the surface of skin lesions)
- More severe than pemphigus foliaceus and pemphigus erythematosus

- Affects moist tissues of the body (mucous membranes), areas where the moist tissues of the body contact the skin, such as the lips (mucocutaneous junctions), and skin; may become generalized
- Ulcers in the mouth are frequent
- Area under the front legs and between the rear legs (known as the “axillae and groin”) often involved
- Positive Nikolsky sign (new or extended erosive lesion created when lateral pressure is applied to the skin near an existing lesion)
- Variable itchiness (pruritus) and pain
- Lack of appetite (known as “anorexia”), depression, and fever
- Secondary bacterial infections are common

Pemphigus Vegetans

- Pustule (raised skin lesion containing pus) groups become masses that ooze
- Involvement of the mouth has not been seen
- No systemic illness

CAUSES

- Unknown

RISK FACTORS

- Unknown



TREATMENT

HEALTH CARE

- Initial inpatient supportive therapy for severely affected patients
- Outpatient treatment with initial frequent hospital visits (every 1 to 3 weeks); taper to every 1 to 3 months when remission is achieved and the patient is on a maintenance medical regimen
- Severely affected patients may need antibiotics and soaks

DIET

- Low-fat—to avoid inflammation of the pancreas (known as “pancreatitis”), which can be a side effect of steroids and (possibly) azathioprine therapy

SURGERY

- Surgical biopsy of the skin lesion and the skin surrounding the lesions



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Pemphigus Foliaceus and Pemphigus Vulgaris

- Steroids—prednisone or prednisolone
- Chemotherapeutic drugs and other drugs to decrease the immune response—more than half of patients require medications other than steroids to decrease the immune response; these drugs generally work in conjunction with prednisone, allowing reduction in dose and side effects of the steroid; examples include azathioprine, chlorambucil, cyclophosphamide, cyclosporine, and dapsone
- Gold-salt treatment or chrysotherapy—gold salts are used to decrease inflammation and the immune response; often used in conjunction with prednisone; include aurothioglucose and auranofin

Pemphigus Erythematous and Pemphigus Vegetans

- Steroids—prednisone or prednisolone administered by mouth
- Steroids administered by application directly to the skin (known as “topical steroids”) may be sufficient in mild cases

Alternative Steroids

- Use instead of prednisone, if undesirable side effects to prednisone or poor response occur

- Methylprednisolone—for patients that tolerate prednisone poorly
- Triamcinolone
- Steroid pulse therapy—methylprednisolone sodium succinate administered intravenously for 3 consecutive days to induce remission; limited application

Topical Steroids (administered to the skin directly)

- Hydrocortisone cream
- More potent topical steroids—0.1% betamethasone, fluocinolone, or 0.1% triamcinonide

Miscellaneous Medications

- Tetracycline and niacinamide



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor response to therapy
- Monitor for medication side effects—routine blood work (complete blood count [CBC] and serum biochemistry), especially patients on high doses of steroids, chemotherapeutic drugs, or gold-salt treatment; check every 1 to 3 weeks, then every 1 to 3 months when in remission

PREVENTIONS AND AVOIDANCE

- Pet should avoid the sun, because ultraviolet (UV) light may worsen the lesions

POSSIBLE COMPLICATIONS

- Depend on type of pemphigus
- Secondary infections
- Side effects of medications may affect quality of life.
- Pemphigus foliaceus and pemphigus vulgaris may be fatal, if untreated (especially pemphigus vulgaris)

EXPECTED COURSE AND PROGNOSIS

Pemphigus Foliaceus and Pemphigus Vulgaris

- Treatment with steroids and chemotherapeutic drugs and medications to decrease the immune response is needed
- Patients may require medication for life
- Monitoring is necessary
- Side effects of medications may affect quality of life
- May be fatal, if untreated (especially pemphigus vulgaris)
- Secondary infections cause morbidity and possible mortality (especially pemphigus vulgaris).

Pemphigus Erythematosus and Vegetans

- Relatively benign and self-limiting
- Steroids administered by mouth eventually may be tapered to low maintenance doses; may be stopped in some patients (as directed by your pet's veterinarian)
- Skin disorder (known as “dermatosis”) develops, if untreated; generalized (systemic) signs are rare
- Prognosis fair



KEY POINTS

- A group of diseases in which the immune-system attacks the skin (known as “autoimmune dermatoses”)
- Pet should avoid the sun, because ultraviolet (UV) light may worsen lesions

PERICARDIAL EFFUSION

(FLUID BUILD-UP BETWEEN THE HEART AND THE SAC SURROUNDING THE HEART)



BASICS

OVERVIEW

- Fluid build-up between the heart and the sac surrounding the heart (known as “pericardial effusion”) with resulting compression of the heart (known as “cardiac tamponade”)
- The sac surrounding the heart is the “pericardial sac” or “pericardium”
- “Effusion” is the medical term for the fluid that builds up within body cavities
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Uncommon in cats

Breed Predilection

- Golden retrievers and German shepherd dogs are more likely than other breeds to have cancer of the right atrium (known as “right atrial hemangiosarcoma”) and to develop fluid build-up of unknown cause (so called “idiopathic pericardial effusion”)
- Short-nosed, flat-faced (known as “brachycephalic”) breeds are more likely than other breeds to develop aortic body tumors (tumors that develop from specialized cells that detect changes in oxygen and carbon dioxide levels in the blood, the cells are located in the aorta [main artery of the body] at the base of the heart)

Mean Age and Range

- Middle-aged to older dogs

Predominant Sex

- Male dogs may be more likely than female dogs to develop fluid build-up of unknown cause (idiopathic pericardial effusion)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Long-term (chronic) fluid build-up between the heart and the sac surrounding the heart (pericardial effusion) often causes fluid build-up in the abdomen (known as “ascites”)
- Sluggishness (lethargy)
- Lack of appetite (known as “anorexia”)
- Weakness
- Exercise intolerance
- Abdominal enlargement or distension
- Breathing distress
- Fainting (known as “syncope”) or collapse

Sudden (Acute) Pericardial Effusion

- Pale gums and moist tissues of the body (known as “mucous membranes”) or the pink color of the gums is slow to return when the gums are blanched by finger pressure (known as “poor capillary refill time”)
- Weak pulses
- Weakness, fainting (syncope), collapse
- Rapid breathing (known as “tachypnea”) and/or rapid heart rate (known as “tachycardia”)

Long-Term (Chronic) Pericardial Effusion

- Enlargement or distension of the jugular veins (located in the neck)
- Fluid build-up in the abdomen (ascites)
- Muffled heart sounds
- Weak pulses
- Pale gums and moist tissues of the body (mucous membranes) or the pink color of the gums is slow to return when the gums are blanched by finger pressure (poor capillary refill time)

- Rapid breathing (tachypnea) and/or rapid heart rate (tachycardia)

CAUSES

- Tumors or cancer (such as hemangiosarcoma), heart-base tumor, tumor or cancer of the thyroid gland (known as “thyroid adenoma” or “adenocarcinoma”), mesothelioma, and cancer that has spread (known as “metastatic cancer”), and lymphoma in cats
- Unknown cause (idiopathic pericardial effusion)
- Blood-clotting disorder (known as a “coagulopathy”)—poisoning with a vitamin K-antagonist rodenticide (such as warfarin); decreased number of platelets in the blood (known as “thrombocytopenia;” “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding); and other blood-clotting disorders
- Infection—feline infectious peritonitis (FIP); fungal infection (coccidioidomycosis); bacterial infection/inflammation of the sac surrounding the heart (known as “pericarditis”)
- Congenital (present at birth) disorders—pericardioperitoneal diaphragmatic hernia (condition in which defects exist in the lower part of the diaphragm [the muscular partition between the chest and abdomen] and the sac surrounding the heart [pericardial sac], allowing abdominal organs [such as the stomach, small intestines, liver] to move into the space between the heart and pericardial sac)
- Tear or trauma to the left atrium or heart
- Congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Foreign body
- Inflammation of the sac (pericardial sac or pericardium) around the heart, characterized by thickening of the sac (condition known as “constrictive pericarditis”) with development of scar tissue



TREATMENT

HEALTH CARE

- Fluid build-up between the heart and the sac surrounding the heart (pericardial effusion) with resulting compression of the heart (cardiac tamponade) warrants immediate tapping and draining of the space between the heart and the sac surrounding the heart (procedure known as “pericardiocentesis”)
- Repeated pericardiocenteses may be needed; surgery may be indicated in selected dogs
- Pericardiocentesis rarely is required in treatment of the cat
- Unless the patient has marked dehydration, administration of fluids generally is not required or recommended for treatment of long-term (chronic) build-up of fluid in the space between the heart and the sac surrounding the heart (pericardial effusion)
- Administer oxygen to dogs with rapid breathing (tachypnea) or signs of circulatory instability

SURGERY

- Surgical removal of part of the sac around the heart (sac is the “pericardium;” procedure is a “pericardectomy”) may be useful in the treatment of fluid build-up between the heart and the sac surrounding the heart (pericardial effusion) accompanying heart-base tumors and may prolong survival
- If fluid build-up is of unknown cause (idiopathic pericardial effusion)—may respond to tapping and draining the space between the heart and the sac surrounding the heart (pericardiocentesis); surgical removal of part of the sac around the heart (pericardectomy) is indicated for recurrent fluid build-up
- Some tumors may be treated surgically



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drugs should not be used in place of tapping and draining the space between the heart and the sac surrounding the heart (pericardiocentesis)
- Medications to remove excess fluid from the body (known as “diuretics”)—may help reduce fluid build-up in the abdomen (ascites), but may lead to side effects and patient weakness
- Vitamin K—indicated for patients with rodenticide-anticoagulant (such as warfarin) poisoning

- Antibiotics are indicated in animals with infection/inflammation of the sac around the heart (infectious pericarditis)
- Chemotherapy—may be useful to treat fluid build-up (effusion) caused by lymphoma; usually ineffective in the treatment of other tumors (such as atrial hemangiosarcoma and heart-base tumor); doxorubicin-based chemotherapy following surgical removal of a tumor involving the right atrium has been shown to increase survival times, but dogs rarely survive more than 6 months following surgery
- Steroids administered by mouth or injection (known as “systemic administration”) or within the sac surrounding the heart (known as “intrapericardial administration”)—may be useful in dogs with fluid build-up of unknown cause (idiopathic pericardial effusion)
- Azathioprine (a chemotherapeutic drug used to decrease the immune response) can be considered for recurrent fluid build-up of unknown cause (idiopathic pericardial effusion)



FOLLOW-UP CARE

PATIENT MONITORING

- Electrocardiogram (“ECG,” a recording of the electrical activity of the heart)—advised during first 24 hours of tapping and draining the space between the heart and the sac surrounding the heart (pericardiocentesis), as the procedure often leads to irregular heart beats (known as “arrhythmias”)
- Fluid build-up between the heart and the sac surrounding the heart (pericardial effusion) may recur at any stage; follow-up examination and performance of an echocardiogram (use of ultrasound to evaluate the heart and major blood vessels) at 10 to 14 days and every 2 to 4 months are recommended to detect recurrent fluid build-up of unknown cause (idiopathic pericardial effusion)

POSSIBLE COMPLICATIONS

- Low blood pressure (known as “hypotension”) or shock
- Air in the space between the lungs and chest wall (known as “pneumothorax”), irregular heart beats (arrhythmias), and tears or cuts into the heart muscle during the procedure to tap and drain the space between the heart and the sac surrounding the heart (pericardiocentesis)

EXPECTED COURSE AND PROGNOSIS

- Right atrial hemangiosarcoma—poor; tumor is highly malignant, usually not able to remove it surgically at time of diagnosis, and it is minimally responsive to chemotherapy
- Chemodectoma—fair; slow-growing tumor, late to spread (metastasize); surgical removal of part of the sac around the heart (pericardectomy) often resolves clinical signs; survival of up to 3 years has been reported following pericardectomy
- Prognosis is good with fluid build-up of unknown cause (idiopathic pericardial effusion); approximately 50% of cases resolve after 1 or 2 procedures to tap and drain the space between the heart and the sac surrounding the heart (pericardiocentesis); surgical removal of part of the sac around the heart (pericardectomy) is often curative in persistent cases



KEY POINTS

- Fluid build-up between the heart and the sac surrounding the heart (pericardial effusion) with resulting compression of the heart (cardiac tamponade) warrants immediate tapping and draining of the space between the heart and the sac surrounding the heart (pericardiocentesis)
- Prognosis is good with fluid build-up of unknown cause (idiopathic pericardial effusion); approximately 50% of cases resolve after 1 or 2 procedures to tap and drain the space between the heart and the sac surrounding the heart (pericardiocentesis); surgical removal of part of the sac around the heart (pericardectomy) is often curative in persistent cases

PERIODONTAL DISEASE



BASICS

OVERVIEW

● Inflammation of the tissues around and supporting the tooth; the tooth support structures include the gum tissue (known as “gingiva”); the cementum and periodontal ligament (the cementum and periodontal ligament attach the tooth to the bone); and the alveolar bone (the bone that surrounds the roots of the tooth); periodontitis (inflammation/infection of the tissues around and supporting the tooth) indicates some degree of periodontal attachment tissue loss (that is, some loss of the structures [cementum, periodontal ligament, alveolar bone] that attach the tooth to the bone)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Swelling of the gum tissue (known as “gingival tissue”)
- Bad breath (known as “halitosis”)
- Redness or fluid build-up (edema) of the gums
- Variable amounts of plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva) and tartar or calculus (mineralized plaque on the tooth surface)
- Gum surfaces bleed easily on contact (for example, during play or physical examination)
- Loose teeth, missing teeth, and exposure of roots of the teeth

CAUSES

- Plaque bacteria (bacteria found in the thin, “sticky” film that builds up on the teeth)

RISK FACTORS

- Toy breeds with crowded teeth
- Dogs that groom themselves—causes hair to be imbedded in the tissue around the teeth (known as the “gingival sulcus”)
- Other debilitating illnesses
- Poor nutritional state



TREATMENT

HEALTH CARE

- Professional cleaning
- Periodontal surgery
- The ultimate goal of periodontal therapy is to control plaque and prevent attachment loss; a willing patient and a client who can provide home care are important considerations in creating a therapy plan

DIET

- Dry food or hard, biscuit-type foods are preferable to soft, sticky foods
- Dental diets, such as Hill’s Prescription Diet® t/d®—specifically indicated to control plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva) and tartar or calculus (mineralized plaque on the tooth surface) in dogs and cats



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics—clindamycin and amoxicillin/clavulanic acid are approved for periodontal disease
- Tetracycline
- Flagyl



FOLLOW-UP CARE

PATIENT MONITORING

- The degree of periodontal disease determines recall interval; some patients are checked weekly, while others can be evaluated every 3 to 6 months

PREVENTIONS AND AVOIDANCE

- Professional dental cleaning and home care are essential for prevention of periodontal disease
- Your pet's veterinarian will discuss home care and available products and will provide instructions for their use

POSSIBLE COMPLICATIONS

- Loss of teeth; loss of bone structure in lower jaw (mandible), leading to shortened lower jaw; tongue protruding from mouth
- Generalized infection in the body
- Possible heart, liver and/or kidney disease



KEY POINTS

- Periodontal disease is the most common infectious disease in dogs and cats
- Periodontal disease can lead to infection in other areas of the body and may cause heart, liver, and or kidney disease
- Professional dental cleaning and home care are essential for prevention of periodontal disease

PERITONITIS

(INFLAMMATION OF THE LINING OF THE ABDOMEN)



BASICS

OVERVIEW

- An inflammatory process involving the lining of the abdominal cavity; the lining of the abdomen is the “peritoneum”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Abdominal pain—localized or generalized throughout the abdomen
- A “praying” position—for relief of pain
- Vomiting is common
- Low blood pressure (known as “hypotension”) and shock—may develop rapidly
- Rapid heart rate (known as “tachycardia”) and a variety of irregular heart beats (known as “arrhythmias”) often are noted
- Fever is not a consistent finding

CAUSES

Primary Inflammation of the Peritoneum (Peritonitis)

- Uncommon
- Results from direct infection through spread of the disease-causing agent (such as bacteria) through the blood stream

Secondary Inflammation of the Peritoneum (Peritonitis)

- Predominant form of peritonitis
- Results from disruption of the abdominal cavity or a hollow abdominal organ, such as the intestine
- Other causes include breakdown of surgical sites; penetrating abdominal wounds; blunt abdominal trauma; inflammation of the pancreas (known as “pancreatitis”); infection and inflammation of the uterus, with accumulation of pus (known as “pyometra”); liver or prostatic abscesses; and rupture of the gallbladder, urinary bladder, or bile duct

RISK FACTORS

- Trauma
- Gastrointestinal surgery
- Undetected abscess of liver, pancreas, prostate, or uterine stump



TREATMENT

HEALTH CARE

- Inpatient care is needed because intensive monitoring is required
- Fluid therapy and antibiotics, administered intravenously
- Potassium and glucose—may need to be supplemented in the intravenous fluids
- The decision to treat the pet medically (drugs only) or surgically is dictated by the cause (if known) of the inflammation of the lining of the abdomen (peritonitis), the pet’s response to initial treatment, and the anticipated cost of treatment; mild cases that seem to respond to medical treatment may not need surgery

ACTIVITY

- Usually limited, as a result of hospitalization and confinement

DIET

- The approach to nutritional support is determined by the circumstances of each individual patient
- Diet is dictated by the cause of the peritonitis, when identified, and any coexistent conditions (such as heart disease)
- Feeding tube, if necessary, may be placed for nutritional support
- Adequate nutrition—essential to optimize outcome

SURGERY

- Most patients will require surgical exploration of the abdomen to clean and remove dead tissue and, if possible, identify and correct any underlying or contributing factor
- Known bacterial contamination or suspected chemical-related inflammation of the lining of the abdomen (peritonitis)—surgical intervention is necessary
- Many animals will die, even with surgical attention
- Following surgery, the abdomen may be closed or may be left open for drainage; the decision to close the abdomen or leave it open is determined by your pet's veterinarian, based on the degree of abdominal contamination, ability to remove all debris, severity of the illness, and anticipation of complications from the presence of pus-forming bacteria



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics—broad-spectrum; when possible, based on bacterial culture and sensitivity testing
- Pending results of bacterial culture and sensitivity testing—try a combination of an aminoglycoside (such as amikacin, gentamicin) and a cephalosporin (such as cefazolin) or a penicillin (such as ampicillin)
- Fluoroquinolones—such as enrofloxacin or orbifloxacin may be substituted for an aminoglycoside, especially if the animal has impaired kidney function
- Medications to control pain (known as “analgesics”)



FOLLOW-UP CARE

PATIENT MONITORING

- Fluid balance, electrolyte balance, acid–base status—monitor closely
- Frequency of monitoring—varies with patient's condition and response to treatment
- Blood work (including a complete blood count [CBC] and serum chemistry profile) and a urinalysis—every 1 to 2 days during periods of intensive monitoring, even in patients that are responding to treatment

PREVENTIONS AND AVOIDANCE

- Prevention—difficult, except when specific risk factors are identified (such as infection and inflammation of the uterus [pyometra])

POSSIBLE COMPLICATIONS

- Abdomen left open to allow peritoneal drainage—abdominal contents may pass through the opening (known as “herniation”)
- Scar tissue
- Death

EXPECTED COURSE AND PROGNOSIS

- Prognosis—depends on rapid identification and successful management of the underlying cause and appropriate follow-up care
- Inflammation of the lining of the abdomen with bacterial infection (known as “septic peritonitis”)—leaving the abdomen open to allow peritoneal drainage may improve survival



KEY POINTS

- If underlying cause is not identified and managed, patient is at risk for complications
- Many animals with inflammation of the lining of the abdomen (peritonitis) will die, even with surgical exploration of the abdomen
- Treatment, extensive monitoring and intensive care may be costly

BRUISING, PETECHIA, ECCHYMOSIS



BASICS

OVERVIEW

- “Bruising” is an injury to the skin in which blood vessels are broken, leading to discoloration of the tissues from the presence of red-blood cells; “petechia” is a small, pinpoint area of bleeding; “ecchymosis” is a bruise or purplish patch under the skin or moist tissues of the body (known as “mucous membranes”), due to bleeding
- Bruises, petechia, or ecchymoses may appear spontaneously or following minimal trauma
- “Thrombocytopenia” is the medical term for low platelet count; “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- “Thrombocytopathy” is the medical term for any bleeding disorder that occurs due to a malfunction of the platelets
- “Clotting factors” are components in the blood involved in the clotting process—the clotting factors are identified by Roman numerals, I through XIII

GENETICS

- Immune-mediated low platelet count in the blood (thrombocytopenia) is suggested to have a genetic basis, because of the high number of cases in cocker spaniels, toy poodles, and Old English sheepdogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Less common in cats

Breed Predispositions

- Doberman pinschers and Scottish terriers are more likely than other breeds to have von Willebrand deficiency; many other breeds have von Willebrand’s disease; von Willebrand’s disease is a primary bleeding defect caused by low levels of von Willebrand’s factor or decreased function of existing von Willebrand’s factor; von Willebrand’s factor is a type of protein that binds to platelets, causing them to crowd or mass together (aggregate) and to adhere to one another to stop bleeding—if the levels of von Willebrand’s factor are low or if the existing von Willebrand’s factor does not function normally, the platelets do not aggregate and adhere to one another and bleeding is not stopped
- Immune-mediated low platelet count in the blood (thrombocytopenia)—cocker spaniels, toy poodles, and Old English sheepdogs
- Certain breeds have recognized specific bleeding disorders that occur due to a malfunction of the platelets (thrombocytopathies), such as the basset hound, spitz, otter hound, Great Pyrenees, American cocker spaniel, boxer, and the Persian cat

Mean Age and Range

- Middle-aged female dogs are at increased risk

Predominant Sex

- Middle-aged female dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Bruising
- Small, pinpoint areas of bleeding (petechiae) in the skin or moist tissues of the body
- Purplish patch under the skin or moist tissues of the body (ecchymosis)
- Other signs based on underlying cause

CAUSES

Low Platelet Count (Thrombocytopenia)

- Immune-mediated thrombocytopenia—unknown cause (so called “idiopathic disease”); drug-induced thrombocytopenia (such as antibiotic-induced thrombocytopenia); secondary to cancer; and infection-induced thrombocytopenia (secondary to viral, rickettsial, bacterial, protozoal or fungal infection)
- Infectious thrombocytopenia, such as seen with ehrlichiosis, Rocky Mountain spotted fever (RMSF), babesiosis, leptospirosis, feline infectious peritonitis (FIP), feline leukemia virus (FeLV) infection, or cytauxzoonosis
- Bone-marrow suppression, leading to low red-blood cell, low white-blood cell, and/or low platelet counts, such as from estrogen toxicity or chemotherapy

- Bone-marrow infiltration of abnormal cells, as seen with certain cancers (such as multiple myeloma or lymphoma)
- Sequestration of platelets in the liver and/or spleen secondary to cancer or twisting of the organ (known as “torsion”)
- Consumption of platelets, such as in disseminated intravascular coagulopathy or “DIC” (a blood-clotting disorder) or recent extensive bleeding, as seen with rodenticide poisoning

Abnormal Function of the Platelets (Thrombocytopathy)

- Congenital (present at birth) or acquired (condition that develops sometime later in life/after birth) disorders affecting platelet adhesion or aggregation

Blood-Vessel (Vascular) Disease

- Inflammation of blood vessels (known as “vasculitis”) secondary to infection, such as with Rocky Mountain spotted fever (RMSF) or feline infectious peritonitis (FIP)
- Immune-mediated inflammation of blood vessels (vasculitis)

Clotting Factor Deficiency

- Usually do not see small, pinpoint areas of bleeding (petechiae) or purple patches (ecchymoses); most common clinical sign seen is bleeding into body cavities and/or joints

RISK FACTORS

- Severe von Willebrand’s disease is seen in German shorthaired pointers, Shetland sheepdogs, Scottish terriers and Chesapeake Bay retrievers; von Willebrand’s disease is a primary bleeding defect caused by low levels of von Willebrand’s factor or decreased function of existing von Willebrand’s factor; von Willebrand’s factor is a type of protein that binds to platelets, causing them to crowd or mass together (aggregate) and to adhere to one another to stop bleeding—if the levels of von Willebrand’s factor are low or if the existing von Willebrand’s factor does not function normally, the platelets do not aggregate and adhere to one another and bleeding is not stopped
- Previous treatment with nonsteroidal anti-inflammatory drugs (NSAIDs)
- Recent vaccination has been suggested as a risk factor for immune-mediated low platelet count (thrombocytopenia)



TREATMENT

HEALTH CARE

- Usually as an inpatient, until a definitive diagnosis has been made
- Discontinue any medications that may alter platelet function (such as aspirin and other nonsteroidal anti-inflammatory drugs [NSAIDs])
- Discontinue medication that is associated with immune-mediated low platelet counts (thrombocytopenia), such as trimethoprim-sulfa in dogs or methimazole in cats
- Maintain fluid volume with a balanced electrolyte solution
- Avoid injections under the skin (known as “subcutaneous injections”) and into the muscle (known as “intramuscular injections”) as well as drawing blood from the jugular vein
- Blood or platelet transfusions may be necessary and life saving before a definitive diagnosis is been made (ensure blood samples are collected prior to transfusion for any diagnostic testing)
- No specific treatment is available for disorders characterized by abnormal function of platelets that are present at birth (known as “congenital thrombocytopathies”), other than desmopressin acetate (DDAVP), which can be used for type I von Willebrand’s disease to help control bleeding
- Acquired (condition that develops sometime later in life/after birth) disorders affecting platelet function (thrombocytopathies)—underlying disease needs to be corrected
- Inflammation of blood vessels (vasculitis)—underlying disease needs to be treated

ACTIVITY

- Minimize activity to reduce risk of even minor trauma



MEDICATIONS

- Depend on underlying diagnosis



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on underlying diagnosis
- Daily platelet count for patients with low platelet counts (thrombocytopenia), until an adequate response is seen

POSSIBLE COMPLICATIONS

- Signs of disease caused by bleeding into the brain, gastrointestinal tract, or other organs
- Shock caused by blood loss
- Death

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying diagnosis

FEARS, PHOBIAS, AND ANXIETIES IN CATS



BASICS

OVERVIEW

- Fear is the feeling of apprehension resulting from the nearness of some situation or object presenting an external threat; the response of the autonomic nervous system prepares the body for “freeze, fight, or flight;” as such, it is a normal behavior, essential for adaptation and survival
- A phobia is a persistent and excessive fear of a specific stimulus, such as a thunderstorm or separation from an attachment figure
- Anxiety is the anticipation of dangers from unknown or imagined origins that results in normal body reactions (known as “physiologic” reactions) associated with fear; anxiety may occur in the aftermath of a fear-producing event or as a result of unrelated environmental changes that are unpredictable

GENETICS

- Genetic component unknown, but possible
- Breed and/or coat color and personality of the cat’s father (paternal personality) have been linked to individual personality traits in cats

SIGNALMENT/DESCRIPTION of ANIMAL

- Any age, sex, or breed of cat

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs of fear or anxiety can vary among individual cats and may vary for an individual cat in relation to different environmental stimuli
- In mild cases of fear or anxiety, the cat may become tense and more reactive to environmental stimuli; some cats may retreat to perceived safe hiding places; cats in a panic can become very aggressive or destructive in their attempts to get away from the thing they fear
- Hiding and avoidance are seen commonly in fearful or anxious cats
- Body postures associated with fearful behavior include ears flattened to the back or to the side of the head, crouched body posture when resting or moving, lowered head, tail tucked alongside the body or held low
- Pupils often are dilated, and the cat may pant, shake, drool, or shed hair
- If the fear is intense, the cat may lose bladder and bowel control and may express its anal sacs
- Vocalizations are usually minimal, unless the cat is showing defensive behavior in response to a perceived threat
- The cat may pace, vocalize, and solicit attention from the owner
- Urine spraying and some types of destructive scratching may be seen in anxious cats
- Generally no abnormalities directly related to fear or anxiety are found on physical examination, unless the cat has injured itself trying to escape or while seeking shelter during its fright

CAUSES

Fearful behavior in cats can be related to the following factors:

- Genetic influences on temperament
- Learned behavior through negative experiences

RISK FACTORS

- Details of the cat’s early life, if known, may indicate a history of poor socialization and environmental exposure or point out possible genetic influences, such as unfriendly parents or feral ancestry
- Early experience and socialization; cats that did not have the chance to be around other cats or around humans during the first few weeks of life are more likely to be uncomfortable and fearful around them
- If the mother and other adult cats, present during the sensitive socialization period for the kittens, showed fearful/avoidance behavior, subsequent social learning may enhance the effect



TREATMENT

HEALTH CARE

- Identify the specific stimulus that provokes the fearful or anxious behavior
- Avoid exposure or close proximity to the fear-producing stimuli, if possible
- Provide ways for the cat itself to manage the situation, by noting its “hideout” preferences and creating a “safe place” for the cat to go to if the situation cannot be avoided
- If the cat must be handled in the presence of the fear-producing stimuli, caution and physical restraint aids (such as cat muzzles, cat bags) should be used to prevent injury to the cat, veterinary clinic personnel, and cat owner
- Desensitization and counterconditioning to the anxiety or fear-provoking stimulus; systematic desensitization is a program of slowly increasing exposure to the object or situation the cat fears; counterconditioning consists of enhancing an internal and external environment counter to one of fear, usually accomplished with food rewards or other pleasurable stimuli (such as playing with toys)
- Address secondary problems (such as strained social interactions) subsequent to defensive aggression directed towards humans or other cats, or elimination problems that may be the result of fears or anxieties
- Litter box location may need to be altered if fearful or anxious behavior is limiting the cat’s access

ACTIVITY

- Normal interactions with owners encouraged, but contact/outgoing behavior should not be forced

DIET

- Normal dietary routine
- Placement of food and water may need to be altered if fearful or anxious behavior is limiting access to these necessities



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medication can be a helpful adjunct to behavioral modification, if the animal’s fearful or anxious behavior is so intense that it interferes with learning or other normal behavioral activities
- Drug classes most often suggested for fearful behavior focus primarily on increasing the available amount of chemicals that transmit signals in the central nervous system (known as “neurotransmitters,” such as serotonin and GABA [gamma aminobutyric acid]); however, levels of other neurotransmitters are affected
- No drugs are approved by the FDA for the treatment of fears, phobias, or anxieties in cats; your veterinarian will discuss the risks and benefits of medical treatment
- Selective Serotonin Reuptake Inhibitors (SSRIs), such as fluoxetine (Prozac®) and paroxetine (Paxil®)
- Tricyclic Antidepressants (TCAs), such as clomipramine and amitriptyline
- Buspirone
- Benzodiazepines, such as alprazolam
- Pheromone therapy (Feliway®, ComfortZone™), initially developed for urine-marking cases, has been used as an aerosol spray and/or room diffuser to calm fearful and anxious cats; apply the product to the environment, not directly to the cat
- Herbal preparations have been suggested for fearful and anxious behaviors in animals; however, no scientific studies have shown benefit in using these preparations in cats with fears, phobias, or anxieties; use of these compounds should be supervised by your pet’s veterinarian



FOLLOW-UP CARE

PATIENT MONITORING

- Frequent follow-up either in person or by telephone is necessary, especially during the first few months of treatment, in order to motivate the client and monitor the effectiveness of any adjunct drug treatment

PREVENTIONS AND AVOIDANCE

- Frequent, positive early exposure to people, places, and things during the first 3 to 9 weeks of a kitten's life may be helpful in avoiding later fear-based reactions; continued positive exposure throughout the first year of life also may be helpful
- Calm interactions and positive associations with fear-producing stimuli may minimize fear-based reactions

POSSIBLE COMPLICATIONS

- Secondary behavior problems may arise or persist after the fearful or anxious behavior has diminished and will need specific treatment

EXPECTED COURSE AND PROGNOSIS

- Animals with shy personalities or poor socialization histories may show minimal response to treatment
- A realistic "end point" of treatment response depends on the animal's background (socialization history, genetic and individual differences in personality), the home situation, and other factors, such as the frequency of natural exposure to fear-producing stimuli
- Medication may help improve response to behavior modification, but not totally eliminate signs of fear, phobia, or anxiety



KEY POINTS

- Identify and clearly describe the cat's body language, behavior and events or situations that consistently trigger fear or anxiety; information about specific triggers associated with fearful or anxious behavior is helpful in setting up a behavioral modification and environmental management program
- Stimuli triggering fearful or anxious responses may be very specific (such as a particular individual, noise or situation) or more generalized (such as all humans, all loud noises)
- Avoid exposure or close proximity to the fear-producing stimuli, if possible
- Provide ways for the cat itself to manage the situation, by noting its "hideout" preferences and creating a "safe place" for the cat to go to if the situation cannot be avoided
- Understand behavioral expectations—animals with shy personalities or poor socialization histories may show a minimal response to treatment
- A realistic "end point" of treatment response depends on the animal's background (socialization history, genetic and individual differences in personality), the home situation, and other confounding factors such as the frequency of natural exposure to fear-producing stimuli
- A reasonable treatment plan involves case-tailored behavioral modification and environmental adjustments

FEARS, PHOBIAS, AND ANXIETIES IN DOGS



BASICS

OVERVIEW

- Fear is the feeling of apprehension resulting from the nearness of some situation, person, or object presenting an external threat; the response of the autonomic nervous system prepares the body for “freeze, fight, or flight;” as such, it is a normal behavior, essential for adaptation and survival; context determines whether fear response is normal or abnormal/inappropriate; most abnormal reactions are learned and can be unlearned with gradual exposure
- A phobia is a persistent and excessive fear of a specific stimulus, such as a thunderstorm; immediate, excessive anxiety response is characteristic; it has been suggested that once a phobic event has been experienced, any event associated with it or the memory of it is sufficient to generate the response; most common phobias are associated with noises (such as thunderstorms or firecrackers)
- Anxiety is the anticipation of future dangers from unknown or imagined origins that results in normal body reactions (known as “physiologic” reactions) associated with fear; most common visible behaviors are elimination (urination and/or passage of bowel movements [defecation]), destruction, and excessive vocalization; separation anxiety is the most common specific anxiety in companion dogs—when alone, the animal exhibits anxiety or excessive distress

GENETICS

- Profound form of fear and withdrawal of unknown cause (so called “idiopathic fear and withdrawal”)—noted in Siberian huskies, German shorthaired pointers, Chesapeake Bay retrievers, Bernese mountain dogs, Great Pyrenees, border collies, and standard poodles among others; appears to be a strong familial (condition that runs in certain families or lines of dogs) component, with likely genetic influence

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Profound form of fear and withdrawal of unknown cause (idiopathic fear and withdrawal)—noted in Siberian huskies, German shorthaired pointers, Chesapeake Bay retrievers, Bernese mountain dogs, Great Pyrenees, border collies, and standard poodles

Mean Age and Range

- Most fears, phobias, and anxieties develop at the onset of social maturity (12 to 36 months of age)
- Profound form of fear and withdrawal of unknown cause (idiopathic fear and withdrawal)—occurs at 8 to 10 months of age
- Old-age onset separation anxiety of unknown cause (so called “idiopathic separation anxiety”)—may be a variant of a decline in thinking, learning, and memory, frequently associated with aging (known as “cognitive dysfunction”); reported in elderly dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Fears and anxieties—variable signs; diagnosis may be made only on the basis of nonspecific signs for which no identifiable stimulus is present
- Mild fears—signs may include trembling, tail tucked, withdrawal, and hiding; reduced activity and passive escape behaviors
- Panic—signs may include active escape behavior; increased, out-of-context, and potentially injurious motor activity
- Classic signs of sympathetic autonomic nervous system activity, including diarrhea that may be diagnosed as inflammatory bowel disease (IBD) or irritable bowel syndrome (IBS); the autonomic nervous system is involved in the control of muscles in the heart, blood vessels, gastrointestinal tract, and other organs; it is composed of two parts—the sympathetic and the parasympathetic parts; the two parts cause opposing responses; for example, the sympathetic nervous system speeds up the heart and causes the blood vessels to constrict or become small while the parasympathetic nervous system slows the heart and causes the blood vessels to expand or dilate
- Physical examination findings usually normal, except for self-induced injuries
- Anxieties—lesions secondary to anxious behavior (such as lick granuloma) may be more common than generally has been appreciated; “lick granuloma” is a thick, firm, oval lesion located on the leg, caused by the dog frequently licking at the site

CAUSES

- Any illness or painful physical condition increases anxiety and contributes to the development of fears, phobias, and anxieties
- Aging changes associated with nervous system changes; infectious disease (primarily viral infections in the central nervous system) and toxic conditions (such as lead poisoning)—may lead to behavioral problems, including fears, phobias, and anxieties
- Fear—from a horrible experience; dog may have been forced into an unfamiliar experience
- Dogs that are deprived of social and environmental exposure until 14 weeks of age may become habitually fearful

- With phobias and panic—may have history of inability to escape or get away from the stimulus causing the phobia and panic (such as being locked in crate)
- Separation anxiety—history of abandonment, multiple owners, rehoming, or prior neglect is common; dog often abandoned/rehomed because of separation anxiety



TREATMENT

HEALTH CARE

- Usually outpatient
- Inpatient—patient with extreme panic and separation anxiety that needs to be protected until anti-anxiety medications reach effective levels (may take days to weeks); patient that must be treated for or protected from physical injury (such as throwing itself from a window); constant daycare, dog-sitting, or inpatient monitoring and stimulation may be best
- Affected dogs respond to some extent to a combination of behavior modification and treatment with anti-anxiety medication
- Control any condition that causes itchiness (such as skin allergies) and/or pain, because itchiness and pain are both related to the nervous system pathways of anxiety and its perception
- Some patients with profound form of fear and withdrawal of unknown cause (idiopathic fear and withdrawal) may need to live in a protected environment with as few social stressors as possible; these animals do not do well in dog shows

Behavior Modification

- Gear toward teaching the dog to relax in a variety of environmental settings
- Avoid reassuring the dog when it is experiencing fear or panic; the dog may interpret this as a reward for its behavior
- Encourage calmness, but do not reinforce the fear reaction
- Remember that not all dogs are calmer when crated; some dogs panic when caged and will injure themselves if forced to be confined
- Absolutely avoid punishment for behavior related to fear, phobia, or anxiety
- Desensitization and counterconditioning—most effective if the fear, phobia, or anxiety is treated early; goal is to decrease the reaction to a specific stimulus (such as being left alone in the dark); “desensitization” is the repeated, controlled exposure to the stimulus that usually causes a fearful or anxious response, in such a way that the dog does not respond with the undesirable response; with repeated efforts, the goal is to decrease the dog’s undesirable response; “counterconditioning” is training the dog to perform a positive behavior in place of the negative behavior (in this case, fear or anxiety)—for example, teaching “sit/stay” and when performed, the dog is rewarded; then when the dog is placed in a situation where it might show the undesirable response, have it “sit/stay”
- Signs involved are subtle; learn to recognize physical signs associated with the fears, phobias, and anxieties

DIET

- Diet modification may be necessary to control itchiness (such as in food allergies) to decrease nervous system stimulation of pathways of itchiness and anxiety



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Anxiety

- Anti-anxiety medications increase central nervous system levels of serotonin—tricyclic antidepressants (TCAs) and selective serotonin reuptake inhibitors (SSRIs); examples include amitriptyline, imipramine, sertraline, and fluoxetine
- Clomipramine—takes 3 to 5 weeks to begin to be effective; best drug if repetitive behavior is the hallmark sign and for separation anxiety involving repetitive barking, motor activity, or elimination
- Treat coexistent signs symptomatically (such as loperamide for diarrhea)
- Most treatment will be long-term, possibly years; treatment duration will depend on number and intensity of signs and duration of condition
- Minimum treatment should be 4 to 6 months

Phobias and True Panic Disorders

- May respond to benzodiazepines; work best if administered before any signs of anxiety, fear, or panic; must be given minimally 30 to 60 minutes before the anticipated stimulus that causes the phobia or panic reaction

- Medications include diazepam, clorazepate, and alprazolam
- Severe separation anxiety (such as dog breaks out of crate or throws itself from windows) and thunderstorm phobia that is accompanied by panic—alprazolam can be used with other medications on an “as-needed” basis



FOLLOW-UP CARE

PATIENT MONITORING

- Long-term (chronic) treatment—blood work (complete blood count [CBC] and serum biochemistry) and urinalysis: as indicated by clinical signs, annually for young patients, and semiannually for older patients; adjust medication dosages accordingly
- Observe for vomiting, gastrointestinal distress, and rapid breathing (known as “tachypnea”)

PREVENTIONS AND AVOIDANCE

- Expose dogs to a variety of social situations and environments when they are young puppies (up to the time they are 14 weeks of age) to decrease the likelihood of fearful behavior; puppies and dogs that were deprived of social and environmental exposure until 14 weeks of age may become habitually fearful, which can be avoided with only a little exposure during this formative time

POSSIBLE COMPLICATIONS

- If left untreated, these disorders are likely to progress

EXPECTED COURSE AND PROGNOSIS

Anxiety

- Most treatment will be long-term, possibly years; treatment duration will depend on number and intensity of signs and duration of condition
- Minimum treatment should be 4 to 6 months



KEY POINTS

- Early treatment with both behavioral modification and medication is key; if left untreated, these disorders are likely to progress
- Gear behavior modification toward teaching the dog to relax in a variety of environmental settings
- Avoid reassuring the dog when it is experiencing fear or panic; the dog may interpret this as a reward for its behavior
- Encourage calmness, but do not reinforce the fear reaction
- Absolutely avoid punishment for behavior related to fear, phobia, or anxiety

PICA AND COPROPHAGIA

(Eating Nonfood Items and Eating Feces)



BASICS

OVERVIEW

- Eating of nonfood items (known as “pica”), including eating of feces or bowel movement (known as “coprophagia”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Oriental breeds of cat tend to eat fabrics
- Nursing dogs frequently eat the feces of their puppies; females, whether intact or spayed, are more likely to exhibit coprophagia

SIGNS/OBSERVED CHANGES in the ANIMAL

- Eating of nonfood items (for example, dogs—rocks and feces; cats—fabrics and plastics)
- Bad breath (halitosis), if problem is coprophagia
- Pale moist tissues of the body (mucous membranes) and weakness if the animal has a low red-blood cell count (anemia)
- Thin body condition if signs are accompanied by abnormal digestion or absorption of food (maldigestion or malabsorption)
- Nervous system signs if behavior caused by neurologic disease

CAUSES

Behavioral Causes

- Nest cleaning
- Displacement activity from unavailable herbivore feces
- Responding to punishment, by removing evidence of soiling
- Imitating owners’ behavior—cleaning the nest
- Compulsive behavior
- Attention-seeking behavior

Medical Causes

- Low levels of digestive enzymes produced by the pancreas (known as “exocrine pancreatic insufficiency”)
- Inflammatory bowel disease
- Excessive number of bacteria in the small intestine (known as “small intestinal bacterial overgrowth”)
- Enlarged esophagus (known as “megaesophagus”) and/or narrowing of a section of the esophagus (known as “esophageal stricture”)
- Intestinal parasitism
- Excessive production of thyroid hormone (known as “hyperthyroidism”)
- Diabetes mellitus
- Excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Dietary deficiencies—unproven
- Drug induced (such as effect of steroids, progestins, phenobarbital)
- Low red-blood cell count (anemia)—iron deficiency, other
- Abnormal digestion or absorption of food (maldigestion or malabsorption; for example, secondary to exocrine pancreatic insufficiency)
- Hunger
- Nervous system disease—primary central nervous system or secondary to abnormal circulation in the liver (portosystemic shunt) in which excessive ammonia builds up in the body, leading to nervous system signs

Drug-induced Causes

- Administration of steroids or progestins frequently leads to increased appetite and excessive eating (polyphagia) in dogs

RISK FACTORS

- Early weaned oriental-breed cats fed low-roughage diets with no access to prey or grass may be at risk for wool eating
- Confinement of dogs in barren yards with no environmental stimulation or enrichment—especially predisposes to coprophagia
- Underlying disease leading to low red-blood cell counts (anemia), abnormal digestion or absorption of food (maldigestion or malabsorption)



TREATMENT

HEALTH CARE

- Varies depending on whether the cause is medical or behavioral
- Treat any underlying disease (such as hormonal problems, gastrointestinal disease, or disorders of the pancreas) and withdraw any drugs that could cause increased appetite (polyphagia)
- Correct any dietary deficiencies
- When no underlying medical cause exists—(1) limit access to nonfood items to prevent animal from eating them; (2) find a safe substitute that the animal can eat; and (3) change the animal's motivation to ingest the nonfood item

TREATMENT of EATING of NONFOOD ITEMS (PICA)

- If pica is attention-getting, muzzle dog to prevent ingestion and ignore attempts to obtain nonfood items
- Fabric chewing in cats can be treated by: (1) removing plastic and woolen clothes from the cat's environment; (2) applying a pungent or bitter taste to objects, which may discourage consumption; (3) feeding a high-roughage diet or tough meat to chew, or providing a garden of grass or catnip to graze on (interactive owner punishment is not recommended and may increase stress and anxiety that underlie compulsive disorders)

TREATMENT of EATING FECES of BOWEL MOVEMENT (COPROPHAGIA)

- Can treat coprophagia in a number of ways—can decrease access to feces by prompt disposal; walk dogs on a leash to facilitate removal from vicinity of feces
- Use a muzzle or head halter on walks; give the dog a food reward when it defecates, thereby counterconditioning it to expect food rather than search for feces; other recommendations, although unsupported by any published data, include feeding a less digestible diet; use of a meat tenderizer or pancreatic enzymes; and sprinkling noxious tasting/smelling substances on feces
- Bitter and hot substances, such as quinine, cayenne pepper, and commercial products (for example, FOR-BID™, Alpar Laboratories, Inc., La Grange, IL) have yielded variable results
- Taste-aversion learning is another potentially effective method; your pet's veterinarian may suggest treating the feces with an agent that makes the pet nauseated for a short period of time and after a few experiences of coprophagy followed by nausea and not feeling well, the dog may learn to avoid eating feces

DIET

- Feed a good quality, balanced diet
- May need high-fiber or roughage diet



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- If the problem is a compulsive disorder, the animal's motivation may be changed by administering psychologically active drugs, such as clomipramine, tricyclic antidepressants (TCAs), or selective serotonin reuptake inhibitors (SSRIs)
- Cyproheptadine



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor and record abnormal eating habits to determine if the animal's pica or coprophagia is decreasing
- Discuss progress in controlling abnormal eating habits with your pet's veterinarian in 10–14 days
- If dietary management changes did not markedly improve the problem, further diagnostic testing and/or medication may be needed

PREVENTIONS AND AVOIDANCE

- Limit access to nonfood items to prevent animal from eating them
- Find a safe substitute that the animal can eat
- Remove plastic and woolen material from the cat's environment
- Apply a pungent or bitter taste to objects, which may discourage consumption

POSSIBLE COMPLICATIONS

- Gastrointestinal complications—foreign bodies, diarrhea, vomiting, bad breath (halitosis)

EXPECTED COURSE AND PROGNOSIS

- Realistic expectations must be understood; changing a behavior that has become a habit is very challenging
- Immediate control of a long-standing problem is unlikely



KEY POINTS

- Ignore the behavior as much as possible and avoid rewarding the behavior
- Abnormal behavior should be evaluated by your veterinarian as soon as possible to determine if a physical cause exists
- Treatment may include behavioral modification and psychologically active drugs if no physical cause is identified
- Realistic expectations must be understood; changing a behavior that has become a habit is very challenging

PLEURAL EFFUSION

(FLUID BUILD-UP IN THE SPACE BETWEEN THE LUNGS AND CHEST WALL)



BASICS

OVERVIEW

- “Pleural” refers to the pleural cavity; the “pleural cavity” is the space between the lungs and chest wall—normally the space is very small, unless fluid builds up in it
- “Effusion” is the medical term for the fluid that builds up within body cavities
- “Pleural effusion” is an abnormal accumulation of fluid within the space between the lungs and chest wall (pleural cavity)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Varies with underlying cause

Mean Age and Range

- Vary with underlying cause

Predominant Sex

- Varies with underlying cause

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on the fluid volume in the space between the lungs and chest wall (pleural cavity), rapidity of fluid accumulation, and the underlying cause
- Difficulty breathing (known as “dyspnea”); breathing often is shallow
- Rapid breathing (known as “tachypnea”)
- Standing with the elbows away from the body in an attempt to increase lung capacity (known as “orthopnea”)
- Open-mouth breathing
- Bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red-blood cells (discoloration known as “cyanosis”)
- Exercise intolerance
- Sluggishness (lethargy)
- Lack of desire to eat (known as “inappetence”)
- Cough
- Muffled or inaudible heart and lung sounds in the lower chest, heard when listening to the chest with a stethoscope

CAUSES

High Hydrostatic Pressure (pressure of blood within the capillaries; as blood flows through the capillaries, hydrostatic pressure causes fluids to leave the blood and enter the tissues)

- Congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Overhydration (excessive fluid in the body)
- Tumors or cancer within the chest

Low Oncotic Pressure (pressure exerted by dissolved compounds in blood plasma that stay within the circulating blood to help maintain circulating blood volume)

- Low levels of albumin (a type of protein) in the blood (known as “hypoalbuminemia”)—occurs in protein-losing enteropathy and nephropathy (conditions in which proteins are lost from the body through the intestines [enteropathy] or kidneys [nephropathy]) and liver disease

Abnormalities of Blood Vessels (Vascular Abnormalities) or Vessels that Transport Lymph (Known as “Lymphatic” Abnormalities)

- Infectious disease—bacterial, viral, or fungal
- Tumors or cancer (such as mediastinal lymphoma; tumor of the thymus [known as a “thymoma”]; mesothelioma; primary lung tumor, and cancer that has spread [known as “metastatic cancer”])

- “Chylothorax” is an accumulation of chyle in the space between the chest wall and lungs (pleural cavity); “chyle” is a milky to slightly yellow fluid composed of lymph and fats taken up from the intestines and eventually transferred to the circulation through the thoracic duct; “lymph” is a watery fluid that contains white-blood cells that travels through lymphatic vessels—it transports lymphocytes (a type of white-blood cell) and fats from the small intestines to the blood stream; the “thoracic duct” is the main lymph vessel of the body—it crosses the chest near the spine, and empties into the venous circulation

- Chylothorax may develop from lymphangiectasia (condition characterized by dilation of the lymphatic vessels resulting from blockage or obstruction of lymphatic vessels); congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs); blockage of the cranial vena cava (main vein that returns blood from the body to the heart); cancer; fungal infections; heartworm disease; defect or tear in the diaphragm (the muscular partition between the chest and abdomen) that allows abdominal contents (such as the liver, stomach, or intestines) to enter the chest (condition known as a “diaphragmatic hernia”); twisting of a lung lobe (known as a “lung-lobe torsion”); or trauma

- Diaphragmatic hernia (defect or tear in the diaphragm [the muscular partition between the chest and abdomen] that allows abdominal contents [such as the liver, stomach, or intestines] to enter the chest)

- Blood in the space between the lungs and chest wall (pleural cavity; condition known as “hemothorax”), such as from trauma, cancer, or a blood-clotting disorder (known as a “coagulopathy”)

- Twisting of a lung lobe (lung-lobe torsion)

- Blood clots to the lungs (known as “pulmonary thromboembolism”)

- Inflammation of the pancreas (known as “pancreatitis”)

RISK FACTORS

- Heart disease
- Trauma



TREATMENT

HEALTH CARE

- First, perform a medical procedure to tap the chest (known as a “thoracocentesis”) and to remove fluid from the space between the lungs and chest wall (pleural cavity) to relieve breathing distress; if the patient is stable after thoracocentesis, outpatient treatment may be possible for some diseases

- Most patients are hospitalized because they require intensive management, such as indwelling chest tubes (for example, in patients with build-up of pus in the space between the lungs and chest wall [condition known as a “pyothorax”] or those that have had chest surgery)

- Preventing further build-up of fluid in the space between the lungs and chest wall (pleural cavity) requires treatment based on a definitive diagnosis

- Placement of a shunt to drain fluid from the space between the lungs and chest wall (pleural cavity) and into the abdominal cavity (shunt known as a “pleuroperitoneal shunt”) may relieve clinical signs in animals with pleural effusion that does not respond to medical treatment

ACTIVITY

- Depends on underlying disease

DIET

- Depends on underlying disease

SURGERY

- Surgery is indicated for management of some causes of fluid build-up in the space between the lungs and chest wall (pleural effusion), such as for tumors/cancer, diaphragmatic hernia repair, lymphangiectasia, foreign-body removal, and twisting of a lung lobe (lung-lobe torsion)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treatment varies with underlying specific disease

- Medications to remove excess fluid from the body (known as “diuretics”) generally are reserved for patients with diseases causing fluid retention and volume overload (such as congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)



FOLLOW-UP CARE

PATIENT MONITORING

- X-ray evaluation is key to assessment of treatment in most patients

PREVENTIONS AND AVOIDANCE

- Avoid trauma, such as being hit by a car

POSSIBLE COMPLICATIONS

- Depend on underlying disease
- Fluid build-up within the lungs as the lungs are re-inflated or re-expanded (known as “re-expansion pulmonary edema”) may develop after fluid in the space between the lungs and chest wall (pleural effusion) is removed
- Death due to breathing compromise

EXPECTED COURSE AND PROGNOSIS

- Vary with underlying cause, but usually guarded to poor
- In a study of 81 cases of fluid build-up in the space between the lungs and chest wall (pleural effusion) in dogs, 25% recovered completely and 33% died during or were euthanized immediately after completing diagnostic evaluation

BACTERIAL PNEUMONIA



BASICS

OVERVIEW

- Inflammation in the lung as a response to disease-causing bacteria, characterized by accumulation of inflammatory cells and fluid in the lung, conducting airways (bronchi and bronchioles), and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged)
- “Pneumonia” is inflammation of the lungs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats; more common in dogs than in cats

Breed Predilection

- Dogs—sporting breeds, hounds, working breeds, and mixed-breed dogs (greater than 12 kg [26 lbs] of body weight)

Mean Age and Range

- Dogs—range, 1 month to 15 years; many cases in puppies less than 1 year of age

Predominant Sex

- Dogs—60% males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Cough
- Fever
- Labored breathing
- Exercise intolerance
- Lack of appetite (known as “anorexia”) and weight loss
- Sluggishness (lethargy)
- Nasal discharge
- Difficult or rapid breathing
- Abnormal breath sounds on listening to the lungs with a stethoscope (known as “auscultation”)—increased intensity or breath sounds over the bronchi; short, rough snapping sounds (known as “crackles”); and squeaking or whistling sounds (known as “wheezes”)
- Dehydration

CAUSES

Dogs

- *Bordetella bronchiseptica* and *Streptococcus zooepidemicus*—primary bacterial cause of pneumonia
- Gram-negative bacteria and *Mycoplasma* predominate in single bacterial and mixed-bacterial infections
- *Escherichia coli*, *Klebsiella pneumoniae*, *Pasteurella multocida*, *Staphylococcus*, *Streptococcus*, *Bordetella bronchiseptica*, *Mycoplasma*, and *Pseudomonas aeruginosa*—most common bacterial isolates
- Anaerobic bacteria (bacteria that can live and grow in the absence of oxygen)—found in lung abscesses and various types of pneumonia (particularly with aspiration or foreign bodies)

Cats

- Bacteria—*Bordetella bronchiseptica*, *Pasteurella*, and *Moraxella* most frequently reported; *Mycoplasma* considered a primary disease-causing microorganism (known as a “pathogen”) in the lower respiratory tract
- Carrier state—may exist; periods of shedding *Bordetella bronchiseptica* after stress; infected female cats (queens) may not shed the organism during pregnancy (prepartum) but begin shedding it after delivering the kittens (postpartum), serving as a source of infection for kittens

RISK FACTORS

- Pre-existing viral infection
- Regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth), dysphagia (difficulty swallowing), or vomiting (forceful ejection of stomach contents up through the esophagus and mouth)
- Functional or structural (anatomic) defects—paralysis of the voice box or larynx (known as “laryngeal paralysis”); enlarged esophagus

(known as “megaesophagus”); cleft palate; inherited disorder in which the normal secretion clearance mechanism of the lungs is defective (known as “primary ciliary dyskinesia”); abnormally small windpipe or trachea (known as “tracheal hypoplasia”)

- Foreign body in the bronchi (part of the airway)
- Reduced level of consciousness—stupor, coma, or anesthesia
- *Mycoplasma*, parasitic, or fungal respiratory infection
- Chest trauma or surgery
- Long-term (chronic) dilation of bronchi or bronchioles, as a consequence of inflammation or blockage of the airway (known as “bronchiectasis”)
- Drugs to decrease the immune response (known as “immunosuppressive drugs”)—such as chemotherapeutic drugs and steroids
- Severe metabolic disorders—excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”); sugar diabetes (diabetes mellitus); excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”) and inadequate production of steroids by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”)
- Presence of pus-forming bacteria and their poisons in the blood or tissues (known as “sepsis”)
- Protein–calorie malnutrition
- Inability to develop a normal immune response (known as “immunodeficiency”)
- Age—very young more susceptible to fatal infections
- Abnormal function of cells that normally remove bacteria and foreign materials from the body (known as “phagocyte dysfunction”)—feline leukemia virus (FeLV) infection and diabetes mellitus
- Complement (a protein substance in the blood that contributes to the destruction and removal of bacteria from the body) deficiency—rare
- Selective immunoglobulin A (IgA) deficiency; immunoglobulin A is an immune protein, found in the intestines; it functions as a protective barrier to prevent limit antigens (substance to which the immune system is responding and producing antibodies) and disease-causing microorganisms from entering the body through the intestines
- Combined T-cell and B-cell dysfunction—rare; a lymphocyte is a type of white-blood cell, formed in lymphatic tissue throughout the body; lymphocytes are further divided into T lymphocytes (which are involved in cell-mediated immunity), so called “T-cells” and B lymphocytes (which produce antibodies as part of the immune process), so called “B-cells”



TREATMENT

HEALTH CARE

- Inpatient—recommended with signs involving multiple body systems (such as lack of appetite [anorexia], high fever, weight loss, and sluggishness [lethargy])
- Maintain normal hydration—important to aid the normal secretion clearance mechanism of the lungs; use a balanced electrolyte solution
- Administration of medication in a fine spray (known as “nebulization”) with saline aerosol—results in more rapid resolution, if used with physiotherapy and antibiotics
- Physiotherapy—mild forced exercise; efforts to dislodge secretions in the lungs and to induce coughing (known as “coupage”); windpipe manipulation to stimulate mild cough; and postural drainage; may enhance clearance of secretions; always do immediately after nebulization; avoid allowing the patient to lie in one position for a prolonged time
- Oxygen therapy—for severe breathing difficulties (known as “respiratory distress”)

ACTIVITY

- Restrict during treatment (inpatient or outpatient), except as part of physiotherapy after administration of medication in a fine spray (nebulization)

DIET

- Ensure normal intake of food, with foods high in protein and calorie or energy density
- Feeding directly into the intestinal tract (known as “enteral feeding”) or through the veins (known as “parenteral nutrition”)—indicated in severely ill patients
- Use caution in feeding animals with an enlarged esophagus (megaesophagus); lack of normal function of the voice box or larynx (known as “laryngeal dysfunction”) or surgery on the voice box or larynx; and disease of the throat or pharynx (known as “pharyngeal disease”)

SURGERY

- Surgical removal of a lung lobe (known as “lung lobectomy”)—may be indicated with lung abscesses or foreign body in the bronchus with secondary pneumonia; may be indicated if patient is unresponsive to conventional treatment and disease is limited to one or two lobes of the lung(s)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antibiotics

- Antibiotics are best selected based on results of bacterial culture and susceptibility testing from transtracheal wash (a technique in which samples from the lower airways are obtained for bacterial culture and/or for evaluation through a microscope) or other diagnostic techniques
- Reasonable initial antibiotic choices pending culture results include amoxicillin–clavulanic acid, cephalexin, chloramphenicol, enrofloxacin, or trimethoprim-sulfonamide
- Gram-positive cocci—ampicillin, ampicillin-sulbactam; amoxicillin; amoxicillin–clavulanic acid; azithromycin; chloramphenicol, erythromycin; gentamicin; trimethoprim-sulfonamide; first-generation cephalosporins
- Gram-negative rods—amikacin; chloramphenicol; gentamicin; trimethoprim-sulfonamide; enrofloxacin; marbofloxacin; carboxypenicillins
- Bordetella*—tetracyclines; amikacin; chloramphenicol; gentamicin; enrofloxacin; kanamycin; azithromycin
- Mycoplasma*—doxycycline, enrofloxacin, marbofloxacin, chloramphenicol
- Anaerobes (bacteria that can live and grow in the absence of oxygen)—amoxicillin–clavulanic acid; chloramphenicol; metronidazole; clindamycin; ticarcillin-clavulanic acid
- Continue treatment for at least 10 days beyond clinical resolution; usually 3 weeks or longer

Expectorants

- Recommended by some veterinarians; no objective evidence that they increase movement of mucus or mobilization of secretions



FOLLOW-UP CARE

PATIENT MONITORING

- Complete blood count (CBC) should be performed periodically; CBC should return to normal as the patient responds to treatment
- Arterial blood gases, to monitor levels of oxygen and carbon dioxide in the blood—most sensitive monitor of progress
- Listen to the patient’s lungs (auscultate) thoroughly several times daily, while hospitalized
- Chest X-rays—improve more slowly than the clinical signs

PREVENTIONS AND AVOIDANCE

- Vaccination—against upper respiratory viruses; against *Bordetella bronchiseptica*, if dog is boarded or exposed to large number of other dogs
- Catteries—environmental strategies to lower the number of cats or the close proximity in which they are housed (known as “population density”) and improve hygiene help control outbreaks of bordetellosis (infection caused by *Bordetella*)

POSSIBLE COMPLICATIONS

- Young dogs infected with *Bordetella bronchiseptica* may develop long-term (chronic) inflammation of the bronchi (bronchitis)

EXPECTED COURSE AND PROGNOSIS

- Prognosis—good with aggressive anti-bacterial and supportive therapy; more guarded in young animals, patients with decreased ability to develop a normal immune response (immunodeficiency), and patients that are debilitated or have severe underlying disease
- Prolonged infection—potential for long-term (chronic) inflammation of the bronchi (bronchitis) or chronic dilation of bronchi or bronchioles, as a consequence of inflammation or blockage of the airway (bronchiectasis) in any patient
- High death rates are associated with severely low levels of oxygen in the blood (known as “hypoxemia”) and presence of pus-forming bacteria and their poisons in the blood or tissues (sepsis)



KEY POINTS

- Inflammation in the lung as a response to disease-causing bacteria, characterized by accumulation of inflammatory cells and fluid in the lung, conducting airways (bronchi and bronchioles), and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged)
- More common in dogs than in cats
- Antibiotics are best selected based on results of bacterial culture and susceptibility testing
- High death rates are associated with severely low levels of oxygen in the blood (known as “hypoxemia”) and presence of pus-forming bacteria and their poisons in the blood or tissues (sepsis)

EOSINOPHILIC PNEUMONIA

(INFLAMMATION OF THE LUNGS WITH EOSINOPHILS, A TYPE OF WHITE-BLOOD CELL)



BASICS

OVERVIEW

- Inflammation in the lung as a response to antigens (substances that induce sensitivity or immune response), characterized by accumulation of eosinophils (a type of white-blood cell) and fluid into the lung, conducting airways (bronchi and bronchioles), and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged)
- “Pneumonia” is inflammation of the lungs
- “Eosinophils” are a type of white-blood cell; they are involved in allergic responses by the body and are active in fighting and damaging larvae of parasites in the body

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilection

- The Siberian husky may be more likely to develop eosinophilic pneumonia than other breeds

Mean Age and Range

- All ages

SIGNS/OBSERVED CHANGES in the ANIMAL

- Extremely variable, depending on the severity
- Harsh, moist cough—unresponsive to antibiotic therapy
- Fever
- Labored breathing
- Difficulty breathing (known as “dyspnea”)
- Exercise intolerance
- Lack of appetite (known as “anorexia”)
- Sluggishness (lethargy)
- Weight loss
- Nasal discharge (may be yellow-green or a combination of mucus and pus)
- Abnormal breath sounds on listening to the lungs with a stethoscope (known as “auscultation”)—increased-intensity breath sounds; short, rough snapping sounds (known as “crackles”); and squeaking or whistling sounds (known as “wheezes”); decreased sounds can occur
- Enlarged lymph nodes (known as “peripheral lymphadenopathy”)—rare

CAUSES

- Substances to which the dog has developed an allergy that are spread through the air (known as “aeroallergens”)—spores or threadlike filaments (known as “hyphae”) from fungi and actinomycetes; pollen; insect antigens; unidentified triggers of the immune response
- Parasitic antigens—heartworm microfilariae (the immature form of the heartworm, found in the blood of animals, especially dogs); respiratory parasites (parasites that reside in the respiratory tract or in the blood vessels of the lungs)

RISK FACTORS

- Living in a heartworm-endemic area (that is, an area where heartworms commonly are found in dogs and to a lesser extent, found in cats), without receiving heartworm preventive medication
- Dusty or moldy environment
- Air pollution



TREATMENT

HEALTH CARE

- Inpatient—recommended with signs involving multiple body systems (such as lack of appetite [anorexia], weight loss, and sluggishness [lethargy])
- Maintain normal hydration—important to aid the normal secretion clearance mechanism of the lungs; use a balanced electrolyte solution
- Oxygen therapy—for severe breathing difficulties (known as “respiratory distress”)

ACTIVITY

- Restricted during treatment (inpatient or outpatient)

DIET

- Ensure normal intake

SURGERY

- May surgically remove lung lobes with large inflammatory nodules (known as “granulomas”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids—prednisolone or prednisone, until clinical signs begin to resolve; then decrease dose slowly (over months), as directed by your pet’s veterinarian
- Heartworm treatment—for heartworm-positive dog; initiate after the patient has been stabilized with steroids and rest
- Itraconazole or ketoconazole are drugs used to treat fungal infections; they are considered to be “antifungal drugs”—may be used with confirmed allergic response to fungal infection of the bronchi or lungs, which is a rare condition; use antifungal drugs only if the fungal infection is confirmed by microscopic evaluation of samples from the lungs or fungal culture
- Hyposensitization—“allergy shots” based on results of skin or serum testing may be attempted to decrease the allergic response, but is not the treatment of choice in most patients; most dogs still will require steroid therapy
- Other drugs that decrease the immune response (known as “immunosuppressive drugs”), such as cyclosporine, cyclophosphamide, azathioprine, and mercaptopurine—may use when steroids are contraindicated or have been ineffective
- Drugs to dilate the bronchi or bronchioles (known as “bronchodilators”)—may be helpful, particularly if squeaking or whistling sounds (wheezes) are heard when listening to the lungs with a stethoscope or labored breathing is observed



FOLLOW-UP CARE

PATIENT MONITORING

- Complete blood count (CBC) will show resolution of increased number of eosinophils in the circulating blood (known as “peripheral eosinophilia”) as the animal responds to treatment
- Arterial blood gases, to monitor levels of oxygen and carbon dioxide in the blood—most sensitive monitor of progress
- Listen to the patient’s lungs (auscultate) thoroughly several times daily, while hospitalized
- Chest X-rays—improve more slowly than the clinical signs

PREVENTIONS AND AVOIDANCE

- Routine heartworm-prevention medication
- Change patient’s environment, if a substances to which the dog has developed an allergy that is spread through the air (aeroallergen) is

suspected

POSSIBLE COMPLICATIONS

- Blood clots in the lungs (known as “pulmonary thromboembolism”)—patients treated for adult heartworms (known as “dirofilariasis”)

EXPECTED COURSE AND PROGNOSIS

- If primary allergen (substance to which the dog has developed an allergy) is identified and eliminated—prognosis good for mild cases
- If allergen (substance to which the dog has developed an allergy) is not identified—prognosis for control good; many patients require long-term treatment with steroids
- Heartworm infection—prognosis depends on severity of increased blood pressure in the lungs (known as “pulmonary hypertension”); enlargement of the right ventricle of the heart (known as “cor pulmonale”); and blood clots (thromboembolism)
- Condition characterized by multiple large, inflammatory nodules containing eosinophils (known as “eosinophilic granulomatosis”)—prognosis guarded; often disease is progressive
- High death rates are associated with severely low levels of oxygen in the blood (known as “hypoxemia”)



KEY POINTS

- Inflammation in the lung as a response to antigens (substances that induce sensitivity or immune response), characterized by accumulation of eosinophils (a type of white-blood cell) and fluid into the lung, conducting airways (bronchi and bronchioles), and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged)
- High death rates are associated with severely low levels of oxygen in the blood (known as “hypoxemia”)

FUNGAL PNEUMONIA



BASICS

OVERVIEW

- Inflammation of the interstitial, lymphatic, and peribronchial tissues of the lung, caused by deep fungal (known as “mycotic”) infection; “interstitial” relates to spaces within tissues or organs; “lymphatic” refers to vessels within the body that transports lymph, a clear to slightly colored liquid that contains white-blood cells—it serves many functions including removing bacteria from tissues and returning fluids to the circulation; “peribronchial” refers to something that surrounds the bronchus or bronchi (airways going from the windpipe [trachea] into the lungs)
- Various fungi can cause “deep fungal infections;” they include *Blastomyces*, *Histoplasma*, *Coccidioidomyces*, *Cryptococcus*; and *Aspergillus*; the fungi are found in different locations in the United States
- Depends on geographic distribution in the United States: Blastomycosis—seen in the Southeast and Midwest, along the Mississippi, Ohio, Missouri, and Tennessee Rivers and southern Great Lakes; also in southern Midatlantic states; Histoplasmosis—similar to, but more widely distributed than, blastomycosis; pockets of disease in Texas, Oklahoma, and California; Coccidioidomycosis—Southwest from Texas to California; Cryptococcosis and Aspergillosis—widespread throughout the United States
- “Pneumonia” is inflammation of the lungs

GENETICS

- Breed susceptibilities may be related to defects in cell-mediated immunity

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and less commonly in cats

Breed Predilection

- Generalized (systemic) disease caused by a fungus (known as “systemic mycosis”)—large-breed dogs kept outdoors or used for hunting or field trials; Doberman pinschers and rottweilers may be susceptible to more severe wide-spread (disseminated) disease
- Generalized (systemic) disease caused by *Aspergillus* (known as “aspergillosis”)—German shepherd dogs may be overrepresented in population of affected dogs

Mean Age And Range

- Young animals (less than 4 years of age) are more susceptible than other ages
- Any age may be affected

Predominant Sex

- Males affected 2 to 4 times more often than females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend primarily on the organ systems involved
- Illness affecting many body systems
- Chronic weight loss and lack of appetite (inappetence)
- Fever
- Discharge from eyes and/or nose
- Coughing—may be prominent; seen inconsistently even with severe lung disease; may be triggered by putting pressure on or feeling the windpipe or trachea
- Difficulty breathing (known as “dyspnea”) or exercise intolerance common; difficulty breathing may be noted when the animal is resting, if severe disease
- Labored breathing—more common in cats; sign of severe disease in both dogs and cats
- Sudden (acute) blindness or squinting of the eyes (known as “blepharospasm”)—if eyes are affected
- Raised bumps (known as “papules”) and nodules on the skin—common, but often missed until draining tracts appear
- Lameness—common if the feet are affected or if inflammation/infection of the bone (known as “osteomyelitis”) develops
- Depression and emaciation—in patients with long-term (chronic) disease
- Fever—about 50% of patients
- Harsh, loud breath sounds—common when listening to the lungs with a stethoscope (known as “auscultation”)
- Short, rough snapping sounds (known as “crackles”) may be heard when listening to the lungs with a stethoscope (auscultation)—may

be prominent, especially in cats

- Blastomycosis—multiple nodules on and under the skin, with draining tracts; inflammation of the iris and other areas in the front part of the eye (known as “uveitis”); loss of attachment of the retina, the back part of the eye, characterized by the presence of multiple nodules (known as “granulomatous retinal detachment”) common
- Coccidioidomycosis (dogs)—severe pain caused by inflammation/infection of the bone (osteomyelitis) common
- Histoplasmosis (dogs)—emaciation and diarrhea (often bloody) prominent
- Cryptococcosis—infection involving the nasal passages and surrounding soft tissue is common

CAUSES

- Blastomyces dermatitidis*—lungs are the primary route of infection
- Histoplasma capsulatum*—lungs and possibly gastrointestinal tract are the primary routes of infection
- Coccidioides immitis*—lungs are the primary route of infection
- Cryptococcus neoformans*—nasal cavity is the primary route of infection, with direct extension into the eyes or central nervous system (brain, spinal cord)
- Aspergillus*—nasal cavity and lungs are the primary routes of infection

RISK FACTORS

- Blastomycosis, histoplasmosis, and cryptococcosis—environmental exposure to soils rich in organic matter; exposure to bird droppings or other fecal matter may make patient susceptible to blastomycosis and cryptococcosis
- Coccidioidomycosis—environmental exposure to sandy, alkaline soil after periods of rainfall; outdoor activities (such as hunting and field trials); decreased ability to develop an immune response (immunosuppression), especially poor cell-mediated immunity, may contribute to generalized (systemic) spread of fungal infection
- Cats—feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) infection
- Prednisone—may worsen the disease
- Chemotherapy
- Cancer involving certain cells in the lymph nodes, spleen, and/or bone marrow (known as “lymphoreticular cancer”)



TREATMENT

HEALTH CARE

- Outpatient—if patient is still eating
- Inpatient evaluation and treatment—if patient is dehydrated, has lack of appetite (anorexia), and has severely low levels of oxygen (known as “severe hypoxia”)
- Administration of fluids, potassium, oxygen, and antibiotics, as needed

ACTIVITY

- Restricted

DIET

- Feed high-protein, calorically dense food
- Histoplasmosis, accompanied by severe gastrointestinal involvement—feed highly digestible food

SURGERY

- Localized, inflammatory nodules (known as “granulomas”) involving the eyes or painful eyes due to secondary glaucoma (in which the pressure within the eye is increased) may require surgical removal of the eye(s)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Itraconazole—drug used to treat fungal infections; it is considered to be an “antifungal drug;” most often used first; must be given with

food

- Fluconazole—antifungal drug; drug of choice for cryptococcosis and patients with central nervous system (brain, spinal cord) or urinary tract involvement
- Lipid-complexed amphotericin B—antifungal drug; administered intravenously (IV)
- Ketoconazole—antifungal drug; may be effective; higher incidence of side effects; longer treatment is necessary; relapse is common
- Amphotericin B—antifungal drug; administered intravenously (IV); may be used in combination with azole drug, such as itraconazole or ketoconazole for severely affected patients
- Amphotericin B—alternative; may give under the skin (subcutaneously) diluted in 0.45% saline/2.5% dextrose solution
- Voriconazole—antifungal drug; used for invasive aspergillosis, in which the deep fungal infection spreads through various tissues of the body



FOLLOW-UP CARE

PATIENT MONITORING

- Liver enzymes—evaluated monthly by blood tests, while patient is on itraconazole, fluconazole, or ketoconazole
- Blood urea nitrogen (BUN) and creatinine—measure before each dose of amphotericin B, to monitor effects on the kidneys
- Chest X-rays—re-evaluate before discontinuing treatment

PREVENTIONS AND AVOIDANCE

- Monitor for signs of recurrence

POSSIBLE COMPLICATIONS

- Blindness is usually permanent
- Kidney failure from treatment with amphotericin B

EXPECTED COURSE AND PROGNOSIS

- Blastomycosis—requires a minimum of 2 months of treatment; 60% to 70% of dogs are cured by treatment with itraconazole; those not cured usually relapse
- Other deep fungal infections—continue until 1 month past remission
- Generalized (systemic) aspergillosis—prognosis not as good as for other fungal causes
- Relapse—may occur up to 1 year after treatment



KEY POINTS

- Less than 70% of dogs and a smaller percentage of cats are likely to respond to treatment
- Treatment is expensive and will probably be necessary for more than 2 months
- Clean environmental areas that have high organic matter or feces (take appropriate precautions to protect yourself from breathing in material in the area; you may want to consult with your physician first)

INTERSTITIAL PNEUMONIA



BASICS

OVERVIEW

- “Interstitial” relates to spaces within tissues or organs; the “interstitium” is the small narrow space or gap between tissues or parts of an organ
- “Pneumonia” is inflammation of the lungs
- Interstitial pneumonia is a form of pneumonia in which the inflammatory process occurs in the walls or interstitium of the alveoli
- The alveolus (plural, alveoli) is the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Canine distemper virus infection—greyhounds, Siberian huskies, Weimaraners, Samoyeds, and Alaskan malamutes
- Interstitial lung disease is recognized most commonly in West Highland white terriers, and possibly white Cairn terriers and bull terriers
- *Pneumocystis carinii* is a protozoa that causes lung disease—miniature dachshunds

Mean Age and Range

- Canine distemper virus infection is most common in puppies 3 to 6 months of age
- Interstitial lung disease is recognized most commonly in middle- to old-age West Highland white terriers
- *Pneumocystis carinii*—miniature dachshunds less than 1 year of age at risk
- Endogenous lipid pneumonia is a type of interstitial pneumonia characterized by the presence of macrophages (types of cells that scavenge and clear bacteria and foreign substances out of body tissues) containing lipids (a group of compounds that contain fats or oils) that originated from breakdown of lung tissue and/or blood; it most commonly affects older cats
- Feline idiopathic pulmonary fibrosis-like conditions are characterized by excessive fibrous or scar-type tissue as part of a reactive process in the lungs; these conditions are of unknown cause and are therefore called “idiopathic;” they typically are seen in middle-aged to older cats
- Toxoplasmosis most commonly affects middle-aged cats

Predominant Sex

- Toxoplasmosis most commonly affects male cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on severity of disease
- Rapid breathing (known as “tachypnea”); coughing; difficulty breathing (known as “dyspnea”); standing with the elbows away from the body in an attempt to increase lung capacity (known as “orthopnea”); bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); open-mouth breathing; exercise intolerance; abnormal breath sounds on listening to the lungs with a stethoscope (known as “auscultation”); possible spitting up blood from the lungs (known as “hemoptysis”)
- Mild fever and discharge from the eyes and/or nose often are present with canine adenovirus-2 infection
- Gastrointestinal signs; fever; discharge from the eyes and/or nose; thickening of the skin (known as “hyperkeratosis”) of the footpads; nervous system deficits; and twitching or contraction of a group of muscles (known as “myoclonus”) may be seen with canine distemper virus infection
- Animals with paraquat toxicity often have vomiting, diarrhea, and ulcers in their mouths and throats; they may produce only small amounts of urine (known as “oliguria”); excessive excitability and nervous system signs may be seen in the early phase of poisoning
- Inflammation of the retina (the back part of the eye, condition known as “retinitis”); inflammation of the iris and other areas in the front part of the eye (known as “uveitis”); nervous system signs, and/or gastrointestinal signs with toxoplasmosis

CAUSES

Congenital (present at birth)

- Bronchiolitis obliterans—organizing pneumonia (“BOOP”) is characterized by inflammation of the bronchioles (small airways) and surrounding tissue; the inflammation partially obliterates or closes the airway; it has been described secondary to an inherited disorder in which the normal secretion clearance mechanism of the lungs is defective (known as “primary ciliary dyskinesia”); defective development of the bronchi (known as “bronchial dysgenesis”) may lead to endogenous lipid pneumonia (a type of interstitial

pneumonia characterized by the presence of macrophages [types of cells that scavenge and clear bacteria and foreign substances out of body tissues] containing lipids [a group of compounds that contain fats or oils] that originated from breakdown of lung tissue and/or blood) in the cat

Metabolic

- Inflammation of the lungs, associated with uremia (excess levels of urea and other nitrogenous waste products in the blood; condition is known as “uremic pneumonitis”), possibly in association with BOOP; liver disease; or inflammation of the pancreas (known as “pancreatitis”) in cats

Cancer

- Cancer may lead to long-term (chronic) dilation of bronchi or bronchioles, as a consequence of inflammation or blockage of the airway (known as “bronchiectasis”) or to BOOP
- Lung cancer (known as “pulmonary carcinoma”) is associated with the development of excessive fibrous or scar-type tissue as part of a reactive process (pulmonary fibrosis) in the lungs of cats

Idiopathic (unknown cause)

- Pulmonary interstitial fibrosis (excessive fibrous or scar-type tissue as part of a reactive process in the lungs) and desquamative interstitial pneumonitis, a condition in which macrophages (types of cells that scavenge and clear bacteria and foreign substances out of body tissues) are found in the alveoli; some cases of endogenous lipid pneumonia; BOOP; primary pulmonary alveolar proteinosis (“PAP;” condition in which the alveoli fill with a protein-containing material)

Inflammatory

- Endogenous lipid pneumonia (a type of interstitial pneumonia characterized by the presence of macrophages [types of cells that scavenge and clear bacteria and foreign substances out of body tissues] containing lipid [a group of compounds that contain fats or oils] that originated from breakdown of lung tissue and/or blood) is seen most commonly in cats with inflammation of the bronchi (known as “bronchitis”) and long-term (chronic) dilation of bronchi or bronchioles, as a consequence of inflammation or blockage of the airway (bronchiectasis) or inflammation of the bronchioles characterized by tissue death (known as “necrotizing bronchiolitis”); pulmonary interstitial fibrosis most likely occurs secondary to immune stimulation and/or immune-complex deposition

Infectious

- Dogs—canine distemper virus, canine adenovirus-2, *Leishmania chagasi*, *Pneumocystis carinii*, *Angiostrongylus vasorum*, *Toxoplasma*
- Cats—*Toxoplasma*, feline immunodeficiency virus (FIV)

Toxic

- Inhalation of dusts, gases, or vapors, thiacetarsemide (drug used in the treatment of heartworm disease), aspiration of petroleum-based products in cats, secondary pulmonary alveolar proteinosis (condition in which the alveoli fill with a protein-containing material), paraquat toxicity, silica dust (leading to “silicosis”), asbestos (leading to “asbestosis”)

Vascular (involving the blood vessels)

- Blood clots to the lungs (thromboembolism); parasite larvae circulating in the lungs (known as “larval migrans”)



TREATMENT

HEALTH CARE

- Inpatient care, oxygen therapy, and monitoring for animals with evidence of severe breathing difficulties (known as “respiratory distress”)
- Minimize exposure to house dust, vapors, chemical fumes, or tobacco smoke
- Humidification of the inspired air (using a nebulizer or vaporizer) to liquefy secretions
- Pulmonary alveolar proteinosis (PAP)—therapeutic flushing of the bronchi, bronchioles, and alveoli (known as “bronchoalveolar lavage”) to remove the protein-containing material

ACTIVITY

- Exercise restriction for animals with increased breathing effort
- Use a harness, rather than a restraint collar

DIET

- Weight loss is indicated, if obese



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a

particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics should be used, as indicated by results of bacterial culture and sensitivities
- Inhaled steroids (such as fluticasone) or drugs to enlarge the bronchi and bronchioles (known as “bronchodilators,” such as terbutaline) can be beneficial in animals that require anti-inflammatory drugs
- Drugs to enlarge the bronchi and bronchioles (bronchodilators) might be helpful: sustained release theophylline, terbutaline
- Angiostrongylus vasorum*—levamisole, with or without aspirin or steroids; alternative therapies include fenbendazole, mebendazole, and ivermectin
- BOOP—steroids (prednisone) have been used with clinical success in one case report
- Idiopathic pulmonary fibrosis—no effective therapy available; may try anti-inflammatory steroid therapy with prednisolone and drugs to enlarge the bronchi and bronchioles (bronchodilators), with or without drugs to control cough (known as “antitussives”) or antibiotics, if indicated
- Leishmania chagasi*—meglumine antimonite or sodium stibogluconate
- Paraquat toxicity—induce vomiting and administer activated charcoal, if recent ingestion is known; supportive care, increase production of urine (known as “diuresis”) using furosemide, a diuretic; oxygen therapy as needed; consider use of drugs to decrease the immune response (known as “immunosuppressive drugs”), such as dexamethasone, cyclophosphamide, nicotinamide, superoxide dismutase, and vitamin A



FOLLOW-UP CARE

PATIENT MONITORING

- Observe response to therapy
- Repeat physical examination and listen to the lungs with a stethoscope (auscultation), chest X-rays, laboratory tests, and arterial blood-gas analysis (to evaluate the levels of oxygen and carbon dioxide in the blood), as indicated

PREVENTIONS AND AVOIDANCE

- Avoid proximity to toxic fumes or paraquat (not legally sold in the United States)
- Vaccinate and deworm animals as recommended
- Appropriate insect control

POSSIBLE COMPLICATIONS

- Secondary lung infections are common with most forms of interstitial pneumonia

EXPECTED COURSE AND PROGNOSIS

- Guarded prognosis with *Pneumocystis carinii*, *Toxoplasma*, *Angiostrongylus vasorum*, canine adenovirus-2, canine distemper virus, *Leishmania chagasi*, and endogenous lipid pneumonia
- Poor long-term prognosis with idiopathic pulmonary fibrosis (mean survival time from the beginning of clinical signs: 17 months)
- Poor prognosis with clinical feline immunodeficiency virus (FIV) infection and uremic pneumonitis
- Paraquat toxicity is commonly fatal in dogs



KEY POINTS

- Interstitial pneumonia is a form of pneumonia in which the inflammatory process occurs in the walls or interstitium of the alveoli
- The alveolus (plural, alveoli) is the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged

PNEUMOTHORAX

(AIR IN THE PLEURAL SPACE, THE SPACE BETWEEN THE CHEST WALL AND THE LUNGS)



BASICS

OVERVIEW

- Air accumulation in the pleural space (the space between the chest wall and the lungs); it is categorized as “traumatic” or “spontaneous” and “closed” or “open”
- “Traumatic” pneumothorax—air accumulates in the pleural space, following some type of trauma (such as being hit by a car)
- “Spontaneous” pneumothorax—air accumulates in the pleural space in the absence of trauma; it is a “closed” pneumothorax; spontaneous pneumothorax is primary if it occurs in the absence of underlying lung disease; secondary if associated with underlying lung disease
- “Closed” pneumothorax—no defects (such as a puncture) in the chest wall
- “Open” pneumothorax—defect (such as a puncture) in the chest wall, resulting in communication of the pleural space (the space between the chest wall and the lungs) with the atmosphere
- “Tension” pneumothorax—air is transferred into the pleural space (the space between the chest wall and the lungs) during inspiration and it becomes trapped, condition creates a one-way transfer of air into the pleural space

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Spontaneous pneumothorax—more common in large, deep-chested dogs; Siberian huskies may be more susceptible than other dog breeds

SIGNS/OBSERVED CHANGES in the ANIMAL

- Traumatic pneumothorax—evidence of recent trauma
- Spontaneous pneumothorax—may have signs of lung disease; usually sudden (acute), but can have a slowly progressive onset
- Rapid breathing (known as “tachypnea”)
- Difficulty breathing (known as “dyspnea”)
- Standing with the elbows away from the body in an attempt to increase lung capacity (known as “orthopnea”)
- Shallow, rapid abdominal breathing common
- Rapid heart rate (known as “tachycardia”)
- Reduced lung sounds near the back—can be difficult to appreciate in animals with severe difficulty breathing

Traumatic Pneumothorax

- Other signs of trauma, including shock
- May or may not have evidence of chest trauma
- Open pneumothorax—obvious chest-wall trauma present
- Pale gums and other moist tissues of the body (mucous membranes); obvious bluish discoloration of the skin and mucous membranes of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”) in severe cases
- Air under the skin (known as “subcutaneous emphysema”) in some cases with air in the mediastinum (the mediastinum is the center portion of the chest that contains the heart and other organs [except for the lungs]; condition is known as “pneumomediastinum”) and/or trauma to the windpipe or trachea

CAUSES

- Traumatic pneumothorax—blunt trauma; penetrating chest injuries; penetrating injuries to the neck; following medical procedure to tap the chest (known as “thoracocentesis”); following surgical incision into the chest (known as “thoracotomy”); perforation of the esophagus (the tube running from the throat to the stomach); injury to the windpipe (trachea) following endotracheal intubation during anesthesia/surgery
- Spontaneous pneumothorax—condition characterized by enlarged or dilated airspaces (known as “bullous emphysema”) is most common cause in dogs; migrating foreign body in the lung; lung cancer, lung abscess; feline asthma; pneumonia; fungal lung nodules (known as “mycotic pulmonary granulomas”); lung disease caused by parasites (such as *Paragonimus*); blister or bubble-like structures in the lungs (known as “pulmonary bullae”); large air-filled or fluid-filled sacs in the lungs (known as “pulmonary blebs”)

RISK FACTORS

- Trauma
- Tapping the chest, to withdraw fluid or air (thoracocentesis)
- Surgical incision into the chest (thoracotomy)
- Overinflating the cuff of endotracheal tube, during anesthesia/surgery
- Lung disease
- Migrating grass awns



TREATMENT

HEALTH CARE

- Inpatient care, until air accumulation has stopped or has stabilized
- Animals with breathing difficulty should have the chest tapped (thoracocentesis) to remove a maximal amount of air
- Provide oxygen therapy, until patient is stabilized
- Pain relief (analgesia) with an opioid-type drug, if significant injuries following trauma
- Tapping the chest (thoracocentesis) can be performed with an intravenous catheter attached to an extension set and stopcock or via a butterfly needle; no need to perform thoracocentesis on animal that is not having breathing difficulties; if large open chest wound—cover as cleanly as possible with airtight bandage (use of sterile lubricant/ointment around periphery of wound); must be accompanied by chest-tube placement; will require surgical closure of chest wound, once animal is stable
- Intravenous fluids required in most cases of trauma, but may not be indicated in cases of spontaneous pneumothorax
- Chest-tube maintenance—ensure all connections are air-tight (cable ties are excellent for securing connections); ensure that tube is attached to animal at two points to reduce chance of inadvertent tube removal; clean tube site and change dressing once daily—do not allow animal to chew at chest tube

ACTIVITY

- Strict rest for at least a week following resolution of pneumothorax, in an effort to minimize the chance of recurrence

SURGERY

- Establishing an opening into the chest with a tube (known as “tube thoracostomy”)—use if unable to stabilize with tapping the chest (thoracocentesis) or repeated thoracocentesis procedures are required for continued pneumothorax (air in the space between the chest and the lungs); chest-tube placement (under local or general anesthesia)—chest tube is passed into pleural space, and a purse-string suture is placed in the skin and the tube is secured with ‘Roman sandal’ suture pattern; chest X-rays should be performed after chest-tube placement
- If air is accumulating rapidly in the space between the chest wall and lungs (pneumothorax)—use continuous chest-tube suction; if pneumothorax is not severe or is resolving—use intermittent chest-tube aspiration
- In emergency situation of life-threatening tension pneumothorax (in which air is transferred into the pleural space [the space between the chest wall and the lungs] during inspiration and becomes trapped, creating one-way transfer of air into the pleural space)—consider emergency surgical incision into the chest (known as “thoracotomy”) to convert problem to an “open pneumothorax;” animal then can be intubated with an endotracheal tube and breathing can be controlled by an assistant or by a ventilator, until the animal is stabilized
- Open traumatic pneumothorax—surgery as soon as patient is stable
- Closed traumatic pneumothorax—rarely requires surgical intervention
- Spontaneous pneumothorax—early surgical intervention recommended in dogs; exploratory surgical incision into the chest (thoracotomy) often performed to allow visualization of the lungs, to determine location of the air leakage
- Evaluation of the space between the chest wall and lungs with an endoscope (a special lighted instrument; procedure known as “thoracoscopy”)—may allow visualization of local lesion; allows instillation of substances to cause formation of scar tissue across the pleural space (the space between the chest wall and the lungs) to eliminate the space where air has been collecting (procedure is known as “pleurodesis”)
- Removal of part or all of a lung may be necessary for localized lesions; traumatic lacerations may be sutured
- Pleurodesis (to cause formation of scar tissue across the pleural space [the space between the chest wall and the lungs] to eliminate the space where air has been collecting) with mechanical abrasion of the pleura (lining of the chest) or instillation of a substance intended to cause inflammation in the pleural space



FOLLOW-UP CARE

PATIENT MONITORING

- Breathing rate—increased rate suggests recurrence of pneumothorax (free air in the space between the chest wall and lungs)
- Repeated chest X-rays to attempt to determine amount of air accumulating
- Pulse oximetry (a means of measuring oxygen levels in blood), if breathing room air can help determine oxygenation status; arterial blood gases (measurements of oxygen and carbon dioxide levels in arterial blood) give the best evaluation of oxygenation status, if lung disease is present
- Rate of air removed from chest through the chest tube

PREVENTIONS AND AVOIDANCE

- Keep pets confined—less likely to be injured

POSSIBLE COMPLICATIONS

- Death from low levels of oxygen in the blood (known as “hypoxemia”) and impairment of the circulation or heart and blood vessels (known as “cardiovascular compromise”)
- Development of fluid build-up in the lungs following re-expansion of the lungs after tapping the chest (thoracocentesis) to remove air (condition known as “re-expansion pulmonary edema”) in cats
- Incorrect placement of chest tube—injury to other organs of the body, such as lung-lobe laceration, heart puncture, diaphragmatic laceration, liver trauma
- Infection of the lining of the chest (known as “pleural infection”) from tapping the chest (thoracocentesis) or presence of the chest drain

EXPECTED COURSE AND PROGNOSIS

- Traumatic pneumothorax—if chest trauma is not severe, the prognosis is good with tapping the chest to remove the air (thoracocentesis) and/or chest-drain placement
- Traumatic pneumothorax—with severe chest trauma, patient can deteriorate despite all efforts to stabilize it, usually because of severe bruising of the lungs and/or bleeding into the lungs
- Spontaneous pneumothorax—prognosis depends on underlying cause; if a single, localized lesion can be removed surgically, prognosis is good; if unable to locate lesion or generalized lung disease is present—prognosis is poor; with surgery, recurrence rate is decreased and the time interval before recurrence is increased



KEY POINTS

- Traumatic pneumothorax—possibility of a chest tube and need for hospitalization; some animals may require surgery
- Spontaneous pneumothorax—recommend early surgical intervention in most cases in dogs; possibility of underlying lung disease that may make resolution challenging and recurrence possible—even with surgical incision into the chest (thoracotomy), the source of the leaking air (pneumothorax) may not be found

PODODERMATITIS

(INFLAMMATION OF SKIN OF THE PAWS)



BASICS

OVERVIEW

- “Podo-” refers to the feet or paws; “dermatitis” is the medical term for inflammation of the skin
- “Pododermatitis” is an inflammatory, multifaceted group of diseases that involves the feet of dogs and, less commonly, cats

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs—common
- Cats—uncommon

Breed Predilections

- Short-coated breeds of dogs—most commonly affected; English bulldogs, Great Danes, basset hounds, mastiffs, bull terriers, boxers, dachshunds, Dalmatians, German shorthaired pointers, and Weimaraners
- Long-coated breeds of dogs—German shepherd dogs, Labrador retrievers, golden retrievers, Irish setters, and Pekingese
- Cats—none

Predominant Sex

- Dogs—male

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary considerably depending on the underlying cause

Pododermatitis in Dogs

- Reddened paws (known as “erythema”)
- Fluid build-up (known as “edema”) of the tissues of the paws
- Small, solid masses (known as “nodules”)
- Thickened, raised, flat-topped areas that are slightly higher than the normal skin (known as “plaques”)
- Variable degrees of loss of the top surface of the skin (known as “erosions” and “ulcers”, based on depth of tissue loss)
- Draining tracts
- Blood blisters
- Discharge from the paws may be blood-tinged or may contain pus
- Dried discharge on the surface of the skin lesion (known as a “crust”)
- Inflammation of soft tissue around the nail (known as “paronychia”)
- Paws may be swollen
- May have hair loss (known as “alopecia”) and may be moist from constant licking
- Paws may be saliva stained (have a rust-colored or brownish staining)
- Paws may be painful and/or itchy (known as “pruritus”)
- Regional lymph nodes may be enlarged
- Thickening of the skin (known as “hyperkeratosis”) of the footpads
- Lameness

Pododermatitis in Cats

- Painful inflammation of soft tissue around the nail (paronychia), involving one or more claws
- Small, solid masses (nodules)
- Loss of the top surface of the skin (ulcers)
- Footpads—commonly involved
- Dried discharge on the surface of the skin lesion (crusts)
- Thickened, raised, flat-topped areas that are slightly higher than the normal skin (plaques)
- Thickening of the skin (hyperkeratosis) of the footpads

- Draining tracts
- Paws may be swollen
- Lameness
- Paws may be painful and/or itchy (pruritus)
- Footpads may have loss of pigment (known as “hypomelanosis”) or may have increased pigment (known as “hypermelanosis”)

CAUSES

Infectious Pododermatitis in Dogs

- Bacterial infections—*Staphylococcus intermedius*, *Pseudomonas*, *Proteus*, *Mycobacterium*, *Nocardia*, or *Actinomyces*
- Fungal infections—dermatophytes (a fungus living on the skin, hair, or nails); sporotrichosis; or deep fungal infections (blastomycosis, cryptococcosis)
- Parasitic infections—demodectic mange in dogs (*Demodex canis*), rhabditic dermatitis (*Pelodera strongyloides*), and hookworms
- Protozoal infections—leishmaniasis

Infectious Pododermatitis in Cats

- Bacterial infections—*Staphylococcus intermedius*, *Pseudomonas*, *Proteus*, *Pasteurella*, *Mycobacterium*, *Nocardia*, or *Actinomyces*
- Fungal infections—dermatophytes (a fungus living on the skin, hair, or nails); sporotrichosis; or deep fungal infections (cryptococcosis)
- Parasitic infections—*Neotrombicula autumnalis*, *Notoedres cati*, or *Demodex*
- Protozoal infections—*Anatrichosoma cutaneum*

Allergic Pododermatitis

- Dogs—atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems); food hypersensitivity; allergic contact dermatitis (inflammation of the skin secondary to contact with some substance to which the animal has an allergic reaction)
- Cats—atopy; rare for flea-allergy dermatitis, food hypersensitivity, or contact dermatitis to involve the paws

Immune-Mediated Pododermatitis

- Dogs—pemphigus foliaceus; systemic lupus erythematosus; erythema multiforme; toxic epidermal necrolysis; inflammation of blood vessels (known as “vasculitis”); cold-agglutinin disease; pemphigus vulgaris; bullous pemphigoid; epidermolysis bullosa acquisita
- Cats—pemphigus foliaceus; systemic lupus erythematosus; erythema multiforme; toxic epidermal necrolysis; inflammation of blood vessels (vasculitis); cold-agglutinin disease; plasma-cell pododermatitis

Hormonal Pododermatitis

- Dogs—decreased levels of thyroid hormone (known as “hypothyroidism”); increased levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); hepatocutaneous syndrome (rare skin condition that develops in patients with liver disease or other metabolic diseases)
- Cats—increased levels of thyroid hormone (known as “hyperthyroidism”); increased levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing’s disease); diabetes mellitus (“sugar diabetes”); hormonal pododermatitis is rare in cats

Cancer

- Dogs—squamous cell carcinomas; melanomas; mast cell tumors; keratoacanthomas; inverted papillomas; eccrine adenocarcinomas
- Cats—papillomas; spinocellular epithelioma; trichoepithelioma; fibrosarcoma; malignant fibrous histiocytoma; metastatic primary adenocarcinoma of the lung; other cancers that have spread (known as “metastatic carcinomas”)
- Higher incidence in cats than in dogs

Environmental Causes

- Dogs—irritant contact dermatitis (inflammation or irritation of the skin secondary to contact with some substance to which the dog comes in contact); trauma; concrete and gravel dog runs; excessive exercise; clipper burn; foreign bodies (such as grass lawns, bristle-like hairs of short-coated dogs); thallium toxicity (a type of heavy metal poisoning)
- Cats—irritant contact dermatitis (inflammation or irritation of the skin secondary to contact with some substance to which the cat comes in contact); foreign bodies; thallium toxicity (a type of heavy metal poisoning)

Miscellaneous

- Dogs—sterile interdigital granulomas (a mass or nodular lesion located between the toes)

RISK FACTORS

- Lifestyle and general husbandry conditions—influence development of inflammation of the skin of the paws (pododermatitis)
- Excess exercise, abrasive or moist housing, poor grooming, and/or lack of preventive medical practice may increase likelihood of developing pododermatitis or worsen the condition



TREATMENT

HEALTH CARE

- Outpatient, unless surgery is indicated
- Foot soaks, hot packing, and/or bandaging may be necessary, depending on cause

ACTIVITY

- Depends on severity of lesions and underlying cause

DIET

- Hypoallergenic diet—if food hypersensitivity or allergy is suspected

SURGERY

- Skin biopsy
- Melanomas and squamous cell carcinomas—very poor prognosis; early diagnosis necessitates surgical removal of the digit, digits, or paw
- Infectious pododermatitis—may benefit from surgical removal of diseased tissue before medical therapy



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Depend on underlying cause and presence of secondary infections
- Medications may include long-term antibiotics, antifungals, steroids, chemotherapeutic agents, hormone-replacement therapy, zinc supplementation, or intravenous amino acids



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on underlying cause and treatment protocol selected

PREVENTIONS AND AVOIDANCE

- Environmental cause—good husbandry and preventive medical practices should avoid recurrence
- Allergic cause—avoid the allergen (environmental or food), if possible; “allergens” are substances to which the animal has developed an allergy

POSSIBLE COMPLICATIONS

- Depend on underlying cause and treatment protocol selected

EXPECTED COURSE AND PROGNOSIS

- Success of therapy depends on finding the underlying cause; often the cause is unknown; even when the cause is known, management can be frustrating due to relapses or expense of treatment
- Often the disease only can be managed and not cured
- Surgical intervention is sometimes the only option



KEY POINTS

- Depend on underlying cause and severity of condition
- Good husbandry and preventive medical practices are necessary
- Pododermatitis will be managed, but not cured, in many cases

EROSIVE, IMMUNE-MEDIATED POLYARTHRITIS



BASICS

OVERVIEW

- “Erosive” refers to “wearing away” or “eating into;” “immune-mediated” refers to a condition caused by the response of the immune system; “polyarthritis” is the medical term for inflammation of several joints
- “Erosive, immune-mediated polyarthritis” is an immune-mediated inflammatory disease of joints that results in wearing away (that is, erosion) of joint cartilage in several joints
- Destruction of bone is evident on X-rays of affected joints

GENETICS

- Not known to be hereditary

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs—idiopathic erosive polyarthritis (erosive inflammation of several joints of unknown cause); erosive polyarthritis of greyhounds
- Cats—feline chronic progressive polyarthritis (long-term, progressive inflammation of several joints, characterized by decreased bone density and formation of new bone in the tissue covering the bone [known as the “periosteum”], with collapse of the spaces between bones in the joint)

Breed Predispositions

- Small- or toy-breed dogs—more susceptible to idiopathic erosive polyarthritis (erosive inflammation of the joint of unknown cause)
- Greyhounds—only breed known to be susceptible to erosive polyarthritis of greyhounds

Mean Age and Range

- Idiopathic erosive polyarthritis (erosive inflammation of the joint of unknown cause) in dogs—young to middle-aged (8 months to 8 years of age)
- Erosive polyarthritis of greyhounds—young greyhounds (3 to 30 months of age) are more susceptible than older greyhounds
- Feline chronic progressive polyarthritis (long-term, progressive inflammation of several joints, characterized by decreased bone density and formation of new bone in the tissue covering the bone [periosteum], with collapse of the spaces between bones in the joint)—onset at 1.5 to 4.5 years of age

Predominant Sex

- Feline chronic progressive polyarthritis (long-term, progressive inflammation of several joints, characterized by decreased bone density and formation of new bone in the tissue covering the bone [periosteum], with collapse of the spaces between bones in the joint)—reported to affect only male cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Dogs and cats—initial symmetric stiffness, especially after rest, or intermittent shifting-leg lameness and swelling of affected joints; “shifting-leg” lameness is characterized by lameness in one leg, then that leg appears to be normal and another leg is involved
- Cats—may have a more subtle onset of signs than seen in dogs
- Usually no history of trauma
- May also see vomiting, diarrhea, lack of appetite (known as “anorexia”), fever, depression, and enlarged lymph nodes (known as “lymphadenopathy”)
- Often cyclic—may appear to respond to antibiotic therapy, but may be undergoing spontaneous remission
- Stiffness of gait; lameness; decreased range of motion; grating detected with joint movement (known as “crepitus”); and joint swelling and pain in one or more joints
- Joint instability, partial dislocation (known as a “subluxation”), or dislocation (known as a “luxation”)—depend on duration of disease
- Lameness—mild weightbearing to more severe non-weightbearing

CAUSES

- Unknown cause (so called “idiopathic disease”)
- Immune-mediated mechanism likely
- *Mycoplasma spumans* (possible cause of erosive polyarthritis of greyhounds)—cultured from one affected greyhound; not isolated in other patients
- Feline leukemia virus (FeLV) and feline syncytium-forming virus (FeSFV)—linked to feline chronic progressive polyarthritis

(long-term, progressive inflammation of several joints, characterized by decreased bone density and formation of new bone in the tissue covering the bone [periosteum], with collapse of the spaces between bones in the joint)



TREATMENT

HEALTH CARE

- Usually outpatient
- Physical therapy—range-of-motion exercises, massage, and swimming; may be indicated for severe disease
- Bandages and/or splints—to prevent further breakdown of the joint; may be indicated for severe disease when pet has compromised ability to walk

ACTIVITY

- Limited to minimize aggravation of clinical signs

DIET

- Weight reduction—to decrease stress placed on affected joints

SURGERY

- Healing rates—may be long and protracted; range of recovery levels
- Surgery—generally not recommended as a good treatment option
- Total hip replacement or surgical removal of the femoral head (the “ball”) of the hip joint (procedure known as “femoral head and neck ostectomy” or “FHO”) may be considered
- Joint fusion (known as “arthrodesis”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Idiopathic Erosive Polyarthritis

- Nonsteroidal anti-inflammatory drugs (NSAIDs) in dogs—unrewarding response
- Steroids (such as prednisone)
- Combination of steroids and chemotherapy drugs (such as cyclophosphamide, azathioprine, 6-mercaptopurine, methotrexate, or leflunomide)
- Remission usually induced by combination chemotherapy within 2 to 16 weeks; determined by resolution of clinical signs and confirmation of normal joint-fluid analysis
- Discontinue chemotherapy drugs 1 to 3 months after remission is achieved
- Maintaining remission—alternate-day steroid (prednisone) treatment generally is successful
- Gold-salt therapy (known as “chrysotherapy”), using aurothiomalate—may alleviate signs

Erosive Polyarthritis of Greyhounds

- Treatment is unrewarding
- Antibiotics, nonsteroidal anti-inflammatory drugs (NSAIDs), steroids, chemotherapy drugs, and medications intended to slow the progression of arthritic changes and protect joint cartilage (known as “chondroprotective drugs”), such as polysulfated glycosaminoglycans—fail to induce remission

Feline Chronic Progressive Polyarthritis

- Treatment may help slow progression
- Combination of steroids (prednisone) and chemotherapy drug (cyclophosphamide)—typically used



FOLLOW-UP CARE

PATIENT MONITORING

- Treatment often is frustrating and requires frequent reevaluation
- Clinical deterioration—requires a change in drug selection or dosage or change in treatment
- Important to try to induce remission; allowing the disease to smolder uncontrolled will increase risk of secondary degenerative joint disease (progressive and permanent deterioration of joint cartilage)

EXPECTED COURSE AND PROGNOSIS

- Progression is likely
- Long-term prognosis is poor
- Cure is not expected; remission is the goal of treatment



KEY POINTS

- Treatment often is frustrating and requires frequent reevaluation
- Poor prognosis for cure and complete resolution
- Progression is likely
- Cure is not expected; remission is the goal of treatment

NON-EROSIVE, IMMUNE-MEDIATED POLYARTHRITIS



BASICS

OVERVIEW

- “Erosive” refers to “wearing away” or “eating into;” “non-erosive” refers to the absence of lesions characterized as “wearing away” or “eating into;” “immune-mediated” refers to a condition caused by the response of the immune system; “polyarthritis” is the medical term for inflammation of several joints
- “Non-erosive, immune-mediated polyarthritis” is an immune-mediated inflammatory disease of joints that *does not* cause wearing away (that is, erosion); includes several identified diseases, such as idiopathic polyarthritis, systemic lupus erythematosus (SLE), polyarthritis associated with long-term (chronic) disease (such as chronic infections or cancer), polyarthritis-polymyositis syndrome, polymyositis syndrome, polyarthritis-meningitis syndrome, polyarthritis nodosa, familial renal amyloidosis in Chinese shar peis, lymphocytic-plasmacytic synovitis, juvenile-onset polyarthritis of Akitas, and the proliferative form of feline chronic progressive polyarthritis

GENETICS

- Not known to be inherited

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Non-erosive, immune-mediated polyarthritis of unknown cause (so called “idiopathic non-erosive, immune-mediated polyarthritis”)—large- (more common) and small-breed dogs; uncommon in cats; seen in German shepherd dogs, Doberman pinschers, retrievers, spaniels, pointers, toy poodles, Lhasa apsos, Yorkshire terriers, and Chihuahuas more frequently than expected compared to other breeds
- Systemic lupus erythematosus (“SLE;” autoimmune disease in which the body attacks its own skin and other organs)—tendency to affect large-breed dogs; collies, German shepherd dogs, poodles, terriers, beagles, and Shetland sheepdogs
- Secondary to administration of sulfa drugs—increased sensitivity in Doberman pinschers
- Polyarthritis-meningitis syndrome (inflammation of several joints [polyarthritis] and inflammation of the membranes covering the brain and spinal cord [known as “meningitis”])—reported in Weimaraners, German shorthaired pointers, boxers, Bernese mountain dogs, beagles, rottweilers, and Akitas
- Amyloidosis (condition in which insoluble proteins [amyloid] are deposited outside the cells in various organs, compromising their normal function) and synovitis (inflammation of the membrane lining the joint)—prominent features of a syndrome affecting young Chinese shar peis
- Juvenile-onset polyarthritis reported in Akitas
- Lymphocytic-plasmacytic synovitis in German shepherd dogs and other large-breed dogs; inflammation of the lining of the joint (synovitis), characterized by the presence of lymphocytes and plasma cells; lymphocytes are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process; plasma cells or plasmacytes are a specialized type of white-blood cell; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease

Mean Age and Range

- Dogs—young to middle-aged

Predominant Sex

- Feline chronic progressive polyarthritis (long-term, progressive inflammation of several joints, characterized by decreased bone density and formation of new bone in the tissue covering the bone [known as the “periosteum”], with collapse of the spaces between bones in the joint)—male cats only

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden (acute) onset; single- or multiple-limb lameness
- Stiffness of gait; decreased range of motion; grating detected with joint movement (known as “crepitus”); and joint swelling and pain in one or more joints
- Mild weightbearing to more severe, non-weightbearing lameness
- Lameness may shift from leg to leg
- Usually no history of trauma
- May see vomiting, diarrhea, lack of appetite (known as “anorexia”), fever, increased urination (known as “polyuria”), or increased

thirst (known as “polydipsia”)

- Signs associated with generalized (systemic) disease or infections (such as infection/inflammation with accumulation of pus in the uterus [known as “pyometra”], infection/inflammation of the prostate [known as “prostatitis”], or bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine [known as “diskospondylitis”]), or cancer
- Often cyclic signs—may appear to respond to antibiotic therapy, but may be undergoing spontaneous remission
- Disease may develop when patient is being treated with sulfur-containing antibiotics

CAUSES

- Unknown for most cases
- Immune-mediated mechanism likely
- Long-term (chronic)—associated with coexistent diseases, such as inflammation of the membranes covering the brain and spinal cord (meningitis), gastrointestinal disease, cancer, urinary tract infection, inflammation of the tissues around and supporting the tooth (known as “periodontitis”), bacterial inflammation of the lining of the heart (known as “endocarditis”), heartworm disease, infection/inflammation with accumulation of pus in the uterus (pyometra), long-term (chronic) infection/inflammation of the middle ear (known as “otitis media”) or of the outer ear (known as “otitis externa”), fungal infections, and long-term (chronic) *Actinomyces* or *Salmonella* infections
- May occur as a side effect of medication in which joint inflammation is secondary to a hypersensitivity reaction to the drug; suspected antibiotics include sulfas, cephalosporins, lincomycin, erythromycin, and penicillins
- Feline leukemia virus (FeLV) and feline syncytium-forming virus (FeSFV)—linked to feline chronic progressive polyarthritis (long-term, progressive inflammation of several joints, characterized by decreased bone density and formation of new bone in the tissue covering the bone [periosteum], with collapse of the spaces between bones in the joint)



TREATMENT

HEALTH CARE

- Usually outpatient
- Physical therapy—range-of-motion exercises and swimming; may be indicated for severe disease
- Bandages and/or splints—to prevent further breakdown of the joint; may be indicated for severe disease when pet has compromised ability to walk

ACTIVITY

- Limited to minimize aggravation of clinical signs

DIET

- Weight reduction—to decrease stress placed on affected joints

SURGERY

- Remove source of infection (such as surgical removal of the uterus in cases with infection/inflammation of the uterus [pyometra]), where applicable—no other therapy may be needed in these cases



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Eliminate underlying causes, if possible—long-term (chronic) disease; discontinue treatment with antibiotics that may lead to polyarthritis
- Typical therapy—initial trial of steroids (such as prednisone); if poor response, then add chemotherapy (such as cyclophosphamide or a thiopurine [azathioprine or 6-mercaptopurine])
- Leflunomide—used to decrease inflammation; may be used in combination with azathioprine, prednisone, and cyclophosphamide
- Maintaining remission—alternate-day steroid therapy (such as prednisone) is generally successful
- Feline chronic progressive polyarthritis (long-term, progressive inflammation of several joints, characterized by decreased bone density and formation of new bone in the tissue covering the bone [periosteum], with collapse of the spaces between bones in the joint)—treatment with prednisone and cyclophosphamide may slow progression of disease



FOLLOW-UP CARE

PATIENT MONITORING

- Clinical deterioration—indicates need for a change in drug selection or dosage

EXPECTED COURSE AND PROGNOSIS

- Complete remission—usually achieved in 2 to 16 weeks; determined by resolution of clinical signs and confirmation of normal joint-fluid analysis
- Recurrence rate—30% to 50% once therapy is discontinued
- Systemic lupus erythematosus (SLE) and feline chronic progressive polyarthritis—progression common; guarded prognosis
- Poor prognosis for cure and complete resolution, if a primary cause is not found



KEY POINTS

- Poor prognosis for cure and complete resolution, if a primary cause (such as a long-term [chronic] infection) is not found and treated successfully

INCREASED NUMBER OF RED-BLOOD CELLS (POLYCYTHEMIA)



BASICS

OVERVIEW

- Blood consists of red-blood cells (the most numerous cells normally), white-blood cells, platelets, and plasma (the liquid portion of blood); each of the blood cells and platelets have ranges for the number of cells or platelets that have been established as being “normal;” in the case of red-blood cells, if the red-blood cell count is lower than the normal limit for the low end of the range, the animal has “anemia” and if the red-blood cell count is higher than the normal limit for the high end of the range, the animal has “polycythemia”
- Polycythemia is caused by an increase in packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood); hemoglobin concentration (hemoglobin is the compound in the red-blood cells that carries oxygen to the tissues of the body); and red-blood cell (RBC) count above the normal ranges
- Polycythemia is classified as “relative,” “transient,” or “absolute”
- Relative polycythemia—develops when a decrease in the liquid portion of the blood (plasma volume) produces a high packed-cell volume (PCV), caused by the cellular portion of the blood being a high percentage of the blood volume as compared to the fluid portion and a “relative” increase in circulating red-blood cells; usually caused by dehydration
- Transient polycythemia—caused by contraction of the spleen, which puts extra red-blood cells into the circulation; this response of the spleen is usually momentary and is a response to stress or release of epinephrine, thus the name “transient polycythemia”
- Absolute polycythemia—characterized by a “true” or “absolute” increase in the number of circulating red-blood cells as a result of an increase in bone-marrow production; either primary or secondary to an increase in the production of erythropoietin (the hormone that stimulates the bone marrow to produce red-blood cells)
- Primary absolute polycythemia (known as “polycythemia rubra vera”)—a bone-marrow disorder characterized by the uncontrolled, but orderly production of an excessive number of mature red-blood cells
- Secondary absolute polycythemia—caused by the appropriate release of erythropoietin (the hormone that stimulates the bone marrow to produce red-blood cells) resulting from long-term (chronic) low levels of oxygen in the blood (known as “hypoxemia”) or by an inappropriate and excessive production of erythropoietin or an erythropoietin-like substance in an animal with normal oxygen levels in the blood

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Short-nosed, flat-faced (known as “brachycephalic”) breeds normally have higher packed cell volumes (PCVs) than do breeds with longer noses
- Large, excitable breeds are prone to contraction of the spleen, and thus to transient polycythemia, where the spleen puts extra red-blood cells into the circulation
- Greyhounds typically have high packed cell volumes (PCVs) as compared to other breeds; normal range for the breed is 50% to 65%

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary with the degree of increased number of red-blood cells (polycythemia)
- Relative polycythemia—dehydration (caused by vomiting, diarrhea, or lack of water intake) and production of only small amounts of urine (known as “oliguria”)
- Transient polycythemia—excitement or vigorous exercise
- Absolute polycythemia—sluggishness (lethargy); lack of appetite (known as “anorexia”); bleeding from the nose and nasal passages (known as “epistaxis” or a “nosebleed”); low exercise tolerance; behavioral change; brick-red or bluish moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red-blood cells (bluish discoloration known as “cyanosis”); sneezing; seizures; or stunted growth
- Primary absolute polycythemia (polycythemia rubra vera)—variable degrees of enlargement of the spleen (known as “splenomegaly”) and of the liver (known as “hepatomegaly”), blood clots (known as “thrombosis”), and bleeding; occasional seizures
- Secondary absolute polycythemia caused by low levels of oxygen in the body tissues (known as “hypoxia”)—signs of long-term (chronic) lung disease or heart disease
- Secondary absolute polycythemia caused by inappropriate erythropoietin secretion—signs associated with cancer or either a kidney or hormonal disorder

CAUSES

- Relative polycythemia (common)—vomiting, diarrhea, diminished water intake, excessive urine production (known as “diuresis”), kidney disease, hyperventilation
- Transient polycythemia—excitement, anxiety, seizures, and restraint
- Primary absolute polycythemia (polycythemia rubra vera)—rare, bone-marrow disorder
- Secondary absolute polycythemia caused by low levels of oxygen in the body tissues (hypoxia)—long-term (chronic) lung disease; heart disease; living at high altitude; short-nosed, flat-faced (brachycephalic) breed conformation; and impairment of kidney blood supply
- Secondary absolute polycythemia caused by inappropriate erythropoietin secretion (rare)—kidney disorder (such as a cyst or tumor); excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); excessive production of thyroid hormone (known as “hyperthyroidism”); cancer



TREATMENT

HEALTH CARE

- Depends on type of polycythemia
- Relative polycythemia—rehydration with intravenous (IV) fluids
- Absolute polycythemia—phlebotomy (procedure in which blood is removed from the body via a vein) recommended to reduce the number of circulating red-blood cells to a packed-cell volume (PCV) of 55%; the amount of blood removed should be replaced with intravenous (IV) fluids to prevent the development of low blood pressure (known as “hypotension”), heart and circulatory collapse (known as “cardiovascular collapse”), and blood clots (thrombosis)
- Primary absolute polycythemia (polycythemia rubra vera)—phlebotomy (procedure in which blood is removed from the body via a vein) and hydroxyurea; frequency of bleeding and medication dosage adjusted to maintain a packed cell volume (PCV) of 55% in dogs and 45% in cats
- Secondary absolute polycythemia caused by low levels of oxygen in the body tissues (hypoxia)—phlebotomy (procedure in which blood is removed from the body via a vein) and hydroxyurea; the high packed cell volume (PCV) is an appropriate compensatory response to the low levels of oxygen by the body; thus phlebotomy may be dangerous; if indicated, remove blood at a slower rate; a higher PCV (60% to 65%) may be necessary to sustain life until the cause of low levels of oxygen in the blood (hypoxemia) is identified and can be corrected
- Secondary absolute polycythemia caused by inappropriate erythropoietin secretion—phlebotomy (procedure in which blood is removed from the body via a vein) and removal of the source of erythropoietin

ACTIVITY

- Depends on type and severity of the polycythemia



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Primary absolute polycythemia (polycythemia rubra vera)—hydroxyurea; also may use chlorambucil or busulfan
- Secondary absolute polycythemia caused by low levels of oxygen in the body tissues (hypoxia)—hydroxyurea



FOLLOW-UP CARE

PATIENT MONITORING

- In severely dehydrated animals, packed cell volume (PCV); total plasma protein (a quick laboratory test that provides general information on the level of protein in the fluid portion of the blood); urine output; and body weight are monitored 2 to 3 times daily until normal hydration is maintained
- Patients being treated for primary absolute polycythemia (polycythemia rubra vera) by chemotherapy—monitor weekly for changes in packed cell volume (PCV), white-blood cell counts, and platelet counts during the initial treatment; then monthly for adjustment of

chemotherapy and periodic phlebotomy (procedure in which blood is removed from the body via a vein)

POSSIBLE COMPLICATIONS

- Increased red-blood cells in the blood leading to sludging of the blood (known as “hyperviscosity”) may occur in patients with absolute polycythemia, especially primary absolute polycythemia (polycythemia rubra vera); hyperviscosity may lead to blood clots (thrombosis), sudden lack of blood supply that leads to death of tissues (known as “infarction”), or bleeding
- Chemotherapy may cause bone-marrow suppression, leading to low red-blood cell and low white-blood cell counts
- Adverse effects of hydroxyurea include loss of cells in the bone marrow (known as “bone-marrow hypoplasia”) with resulting low platelet count (known as “thrombocytopenia”) and low neutrophil count (a neutrophil is a type of white-blood cell; condition is known as “neutropenia”); hair loss (known as “alopecia”); changes in skin pigmentation; and sloughing of toe nails; hydroxyurea also may decrease or stop the production of sperm

EXPECTED COURSE AND PROGNOSIS

- Prognosis in primary absolute polycythemia (polycythemia rubra vera) is guarded, but is influenced by the animal’s response to chemotherapy and phlebotomy (procedure in which blood is removed from the body via a vein)
- Secondary absolute polycythemia caused by low levels of oxygen in the body tissues (hypoxia) depends upon the cause of the low levels of oxygen
- Prognosis in secondary absolute polycythemia caused by inappropriate erythropoietin secretion is determined by identification and elimination of the source of erythropoietin



KEY POINTS

- Identification of the cause or mechanisms responsible for the increase in the number of circulating red-blood cells is the major focus in the clinical diagnosis and treatment of polycythemia
- Polycythemia is caused by an increase in packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood); hemoglobin concentration (hemoglobin is the compound in the red-blood cells that carries oxygen to the tissues of the body); and red-blood cell (RBC) count above the normal ranges
- Polycythemia is classified as “relative,” “transient,” or “absolute”

PERIPHERAL NEUROPATHIES (POLYNEUROPATHIES)

(DISORDERS OF THE PERIPHERAL NERVES)



BASICS

OVERVIEW

- “Neuropathy” is the medical term for any disorder affecting the nervous system; “peripheral neuropathy” is a general term for disorders affecting the peripheral nerves; the “peripheral nerves” are the nerves outside of the central nervous system (that is, outside of the brain and spinal cord)—these nerves extend to the head, body, and legs; “polyneuropathy” is a disease or disorder that involves several peripheral nerves
- “Peripheral neuropathies” are diseases or disorders that affect many peripheral motor, sensory, autonomic, and/or cranial nerves, in any combination; “motor nerves” control muscles; “sensory nerves” carry impulses to the central nervous system, so the animal is “aware” of a particular sensation, such as taste or feeling touch or pain; the “autonomic nerves” are involved in the control of muscles in the heart, blood vessels, gastrointestinal tract, and other organs; the “cranial nerves” are nerves that originate in the brain and go to various structures of the head (such as the eye, face, and tongue)
- “Axonopathy” is a disorder affecting the axons of peripheral nerves; the “axon” is the part of the nerve that carries impulses away from the nerve cell body and toward other nerves or toward muscles

GENETICS

- Most inherited as autosomal recessive disorders
- Disorder characterized by loss of nerve cell bodies in the brain stem and spinal cord (known as “spinal muscular atrophy”) in Brittanys—autosomal dominant disorder

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

Inherited Disorders

Spinal Muscular Atrophy (disorder characterized by loss of nerve cell bodies in the brain stem and spinal cord [spinal muscular atrophy])

- Brittanys, Swedish Lapland dogs, English pointers, German shepherds, rottweilers
- Progressive neuronopathy—cairn terriers
- Disorders Affecting the Axons of Peripheral Nerves (Known as “Axonopathies”)*
- Giant axonal neuropathy—German shepherd dogs
- Progressive axonopathy—boxers, Leonbergers
- Presence of large amounts of oxalic acid or oxalates in the urine (known as “primary hyperoxaluria”)—domestic shorthairs (cat breed)
- Paralysis of the voice box or larynx (known as “laryngeal paralysis”)—polyneuropathy complex—Dalmatians, rottweilers and Great Pyrenees (Pyrenean Mountains dogs)
- Distal polyneuropathy—Birman (cat breed)

- Distal sensorimotor polyneuropathy—rottweilers, Alaskan malamutes

Demyelination (disorder characterized by loss of the white material [known as “myelin”] that covers certain nerve fibers)

- Hypertrophic neuropathy—Tibetan mastiffs

Lysosomal Storage Diseases (inherited metabolic diseases in which harmful levels of materials accumulate in the body’s cells and tissues)

- Globoid cell leukodystrophy—West Highland white terriers, cairn terriers, domestic shorthair kittens
- a-L-fucosidosis—English springer spaniels
- G_{M1} gangliosidosis type II—Siamese and mixed-breed cats
- Sphingomyelinosis—Siamese (cat breed)

- Ceroid lipofuscinosis—English setters, Chihuahuas, and Siamese

Sensory Neuropathy (disorder involving sensory nerves)

- Long-haired dachshunds, English pointers, German shorthaired pointers, English springer spaniels, French spaniels and border collies

Acquired (condition that develops sometime later in life/after birth) Disorders

- Coonhound paralysis—because of their use, coonhounds have a higher incidence than other breeds
- Clinical diabetic polyneuropathy—more common in cats than dogs

- Tumor of the pancreas involving the cells that secrete insulin (known as an “insulinoma”)—German shepherd dogs, boxers, Irish setters, standard poodles, and collies

Mean Age and Range

Inherited Disorders

- Usually begin at less than 6 months of age
- High levels of chylomicrons (lipid droplets containing cholesterol esters and triglycerides) in the blood (known as “hyperchylomicronemia”) in cats—usually over 8 months of age
- Presence of large amounts of oxalic acid or oxalates in the urine (known as “primary hyperoxaluria”) in cats—5 to 9 months of age
- Rottweiler distal polyneuropathy—over 1 year of age
- Giant axonal neuropathy in German shepherd dogs—14 to 16 months
- Intermediate and long-term (chronic) forms of spinal muscular atrophy in heterozygote Brittanys—6 to 12 months of age

Acquired (condition that develops sometime later in life/after birth) Disorders

- Secondary to cancer and pancreatic tumors secreting insulin (insulinoma)-associated low blood glucose (known as “hypoglycemia”)—tend to occur in middle-aged and old animals
- *Neospora* inflammation of several nerve roots and nerves (known as “*Neospora* polyradiculoneuritis”)—most commonly seen in dogs less than 6 months of age; highest incidence, 2 to 4 months of age

SIGNS/OBSERVED CHANGES in the ANIMAL

Inherited Disorders

- Most—slow, progressive; generalized weakness, muscle tremors, loss of muscle mass (known as “muscle atrophy”), often with an abnormal stance and gait
- Sensory neuropathies—may see self-mutilation or a wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”)
- Lysosomal storage diseases (inherited metabolic diseases in which harmful levels of materials accumulate in the body’s cells and tissues)—evidence of slowly progressive central nervous system involvement is common; head tremors, wobbly, incoordinated or “drunken” appearing gait or movement (ataxia), seizures, blindness, dementia, and depression
- Giant axonal neuropathy of German shepherd dogs—rapidly progressive (less than 3 weeks) generalized weakness

Acquired (condition that develops sometime later in life/after birth) Disorders

- Rapid or slow progression
- Rapidly progressive course—an initial stiff, stilted gait, leading to progressive generalized weakness or partial paralysis (known as “paresis”) or paralysis (coonhound paralysis, distal denervating disease)
- Slowly progressive course—generalized weakness and loss of muscle mass (muscle atrophy); in the distal polyneuropathies (diabetic cat), an abnormal stance
- Abnormal function of the autonomic nervous system (known as “dysautonomia”)—primarily a sudden (acute) onset (less than 48 hours) of depression, lack of appetite (known as “anorexia”), constipation, third eyelid protrusion, vomiting, and lack of control of urination (known as “urinary incontinence”)
- May see weakness of all four legs (known as “tetraparesis”) to paralysis of all four legs (known as “tetraplegia”); may see decreased reflexes (known as “hyporeflexia”) to lack of reflexes (known as “areflexia”); may see decreased muscle tone (known as “hypotonia”) to lack of muscle tone (known as “atonia”); loss of muscle mass (muscle atrophy); muscle tremors are common
- May see proprioceptive abnormalities, in which the normal subconscious awareness of the location of the limbs and movement is altered
- May have a change in voice (known as “dysphonia”) or loss of voice (known as “aphonia”)
- Other signs determined by underlying cause of the nerve disorder

CAUSES

Acquired (condition that develops sometime later in life/after birth) Disorders

- Immune-mediated disease—primary or secondary; may be seen with systemic lupus erythematosus or other immune-mediated diseases (such as polymyositis, glomerulonephritis, polyarthritis, and pemphigus)
- Metabolic disease—diabetes mellitus (sugar diabetes) in cats; decreased levels of thyroid hormone (known as “hypothyroidism”); and insulinoma; may be associated with various cancers (such as carcinomas, malignant melanoma, mast cell tumor, osteosarcoma, multiple myeloma, or lymphoma)
- Infectious disease—*Neospora caninum*; feline leukemia virus (FeLV)-related disease
- Chemotherapeutic drugs—vincristine; vinblastine; cisplatin; colchicine
- Poisons—thallium; organophosphates; carbon tetrachloride; lindane
- Unknown cause (so called “idiopathic” disease)

RISK FACTORS

- Development of associated specific diseases (metabolic, immune-mediated, cancer) or exposure to associated specific drugs/poisons or causal factors (such as raccoon saliva)



TREATMENT

HEALTH CARE

- Inherited disease—most are untreatable
- Acquired (condition that develops sometime later in life/after birth) disease—principal goal usually is to treat the primary cause, if identified, with the hope that the secondary polyneuropathy will improve or resolve after appropriate therapy; not always successful
- Usually outpatient
- Inpatient—observe sudden (acute) disease of several nerve roots and nerves (polyradiculoneuropathies) closely for breathing failure in the early progressive phase of disease
- Physical therapy—excellent ancillary treatment
- Systemic lupus erythematosus-related disease—treat as for long-term (chronic) progressive or relapsing polyneuropathy
- Abnormal function of the autonomic nervous system (dysautonomia)—may require intensive intravenous fluid therapy and/or feeding

ACTIVITY

- No restrictions, if able to walk (known as being “ambulatory”)

DIET

- Generally no special management, unless the animal has an enlarged esophagus (the tube running from the throat to the stomach; condition known as “megaesophagus”) or difficulty swallowing (known as “dysphagia”)
- High levels of chylomicrons (lipid droplets containing cholesterol esters and triglycerides) in the blood (known as “hyperchylomicronemia”)—low-fat diet alone can resolve the polyneuropathy within 2 to 3 months
- Inability to move (paralysis)—make sure the pet can reach food and water
- Regurgitation (passive, backward movement or return of food or other contents from the esophagus) and/or vomiting (forceful ejection of stomach contents up through the esophagus and mouth), such as in cases with abnormal function of the autonomic nervous system (dysautonomia)—temporarily halt intake of food and/or water
- Diabetes mellitus (“sugar diabetes”)—important to carefully monitor food intake

SURGERY

- If the nervous system disorder is secondary to a tumor or cancer, treat the primary tumor via surgery, chemotherapy, or radiation



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Long-term (chronic) progressive or relapsing disease—most likely immune-mediated; may improve with long-term steroid treatment, designed to decrease the immune response (known as “immunosuppressive steroid therapy”), prednisone may be administered at doses to decrease the immune response; chemotherapeutic drugs may be used to decrease the immune response, examples include azathioprine or cyclophosphamide; response of individual patient is variable
- Cancer—steroids administered to decrease the immune response (immunosuppressive steroid therapy) may improve the polyneuropathy, without specific action against the primary tumor
- Neospora*-associated inflammation of several nerve roots and nerves (polyradiculoneuritis)—best treated with clindamycin; effectiveness is questionable
- Abnormal function of the autonomic nervous system (dysautonomia)—treat symptomatically with intravenous fluid therapy, artificial tears, metoclopramide, bethanechol, and physostigmine eye drops



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat nervous system examinations

PREVENTIONS AND AVOIDANCE

- Avoid breeding animals with inherited or *Neospora*-associated (placental transfer of the organism from the bitch) diseases
- Avoid contact with raccoons for dogs with a previous history of coonhound paralysis

POSSIBLE COMPLICATIONS

- Inherited disease—continued nervous system deterioration, eventually leading to inability to walk (ambulate) successfully
- Sudden (acute) or long-term (chronic) progressive disease—severe loss of muscle mass (muscle atrophy) and resultant pressure sores; urinary tract infection; scarring and contraction of muscles; aspiration pneumonia

EXPECTED COURSE AND PROGNOSIS

- Conditions in which only myelin (a white material that covers certain nerve fibers) is lost (known as “demyelinating disease”) have a more rapid course of improvement than those involving actual loss of function of the axons (known as “axonal degeneration”)—the majority of cases, which can take months for partial or complete recovery, if at all
- Inherited disease—most have a poor to hopeless prognosis for any recovery of peripheral nerve function (except for cats with high levels of chylomicrons [lipid droplets containing cholesterol esters and triglycerides] in the blood [hyperchylomiconemia])
- Sudden (acute) inflammation of several nerve roots and nerves (polyradiculoneuritis or coonhound paralysis)—good long-term prognosis; may take weeks to months to recover ability to walk (ambulation)
- Metabolic disease—fair to good prognosis with successful treatment of the primary metabolic abnormality; tumors of the pancreas involving the cells that secrete insulin (insulinomas) have a high recurrence rate
- Other acquired (condition that develops sometime later in life/after birth) diseases—most show continued deterioration despite treatment; guarded to poor prognosis; sometimes progression is slow and insidious over many months or years



KEY POINTS

- Treatment of the primary cause may not lead to reversal of the peripheral nerve signs, and, in some cases, deterioration will continue
- Many polyneuropathies are of unknown cause (so called “idiopathic disease”)

INCREASED URINATION (POLYURIA) AND INCREASED THIRST (POLYDIPSIA)



BASICS

OVERVIEW

- Polyuria—increased urination; greater than normal urine production (dogs, more than 45 ml of urine per kilogram body weight per day; cats, more than 40 ml of urine per kilogram body weight per day)
- Polydipsia—increased thirst; greater than normal water consumption (dogs, more than 90 ml of water ingested per kilogram body weight per day; cats, more than 45 ml of water ingested per kilogram body weight per day)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Congenital (present at birth) diseases that cause increased urination (polyuria) and increased thirst (polydipsia), such as diabetes insipidus (“water diabetes”); portosystemic shunt (condition in which blood vessels allow blood to flow abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver); and certain kidney diseases; as well as decreased levels of steroids produced by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”); and some causes in which the dog appears to have a “psychological” drive to drink excessive amounts of water (known as “primary polydipsia”)—predominantly affect young dogs
- Kidney failure; increased levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); increased levels of thyroid hormone (known as “hyperthyroidism”); and tumor disorders affecting the pituitary gland and hypothalamus—predominantly affect middle-aged and older dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Increased urination
- Increased thirst
- Other signs determined by underlying disease

CAUSES

- Primary increased urination (polyuria) due to impaired response of the kidneys to antidiuretic hormone (ADH), the hormone that decreases the amount of water in the urine and thus, maintains hydration of the body—kidney failure; increased levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing’s disease) in dogs; increased levels of thyroid hormone (hyperthyroidism) in cats; bacterial infection/inflammation of the kidney (known as “pyelonephritis”); leptospirosis; decreased levels of steroids produced by the adrenal glands (hypoadrenocorticism or Addison’s disease); inflammation with accumulation of pus in the uterus (known as “pyometra”); liver failure; increased levels of calcium in the blood (known as “hypercalcemia”); decreased levels of potassium in the blood (known as “hypokalemia”); abnormalities in the kidney’s ability to concentrate urine; dietary protein restriction; drugs; congenital (present at birth) diabetes insipidus (“water diabetes”)
- Primary increased urination (polyuria) caused by increased production of urine due to the presence of certain substances in the kidney tubules, such as the presence of glucose (sugar; condition known as “osmotic diuresis”)—diabetes mellitus (“sugar diabetes”); kidney problem that allows glucose or sugar to enter the urine, without increased blood glucose levels (known as “primary renal glucosuria”); increased production of urine as the body’s response following relief of blockage or obstruction of urination (known as “postobstructive diuresis”); some medications that remove excess fluids from the body (known as “diuretics”), such as mannitol and furosemide; ingestion or administration of large quantities of dissolve substances (known as “solutes,” such as salt [sodium chloride] or glucose); and condition caused by excessive levels of growth hormone, leading to enlargement of bone and soft-tissues in the body (known as “acromegaly” or “hypersomatotropism”)
- Primary increased urination (polyuria) due to deficiency of antidiuretic hormone (ADH), the hormone that decreases the amount of water in the urine and thus, maintains hydration of the body—unknown cause (so called “idiopathic disease”); trauma; cancer; or congenital (present at birth) diabetes insipidus (“water diabetes”); some drugs (such as alcohol and phenytoin)
- Primary increased thirst (polydipsia)—behavioral problem; fever; pain; or disease of the anterior hypothalamic thirst center of cancerous, traumatic, or inflammatory origin

RISK FACTORS

- Kidney disease or liver disease
- Certain hormonal and electrolyte disorders
- Administration of medications to remove excess fluids from the body (diuretics), steroids, and medications to control seizures (known

as “anticonvulsants”)

- Low-protein diets designed for dissolving of struvite urinary tract stones (known as “uroliths”) in dogs
- Young, hyperactive, large-breed dogs appear to be at higher than normal risk for “psychological” drive to drink excessive amounts of water (primary polydipsia)



TREATMENT

HEALTH CARE

- Serious medical consequences for the patient are rare, if patient has free access to water and is willing and able to drink; until the mechanism of increased urination (polyuria) is understood, access to water should not be limited to avoid possible dehydration (any limitation of access to water should be considered only under the direction of your pet’s veterinarian)
- Direct treatment at the underlying cause
- Provide free access to water, unless the pet is vomiting
- If vomiting, give replacement maintenance fluids via injection (fluids administered intravenously [IV] or under the skin [subcutaneously or SC])
- Provide fluids via injection (fluids administered intravenously [IV] or under the skin [subcutaneously or SC]) when other conditions limit intake of fluids by mouth (oral route) or dehydration persists, despite increased thirst (polydipsia)
- “Psychological” drive to drink excessive amounts of water (primary polydipsia)—treat by gradually limiting water intake to a normal daily volume, as directed by your pet’s veterinarian—it may be necessary to reduce water intake over days to weeks to avoid undesirable behaviors (such as increased barking); monitor closely to avoid dehydration

DIET

- Depends on underlying cause of increased urination (polyuria) and increased thirst (polydipsia)



MEDICATIONS

- Vary with underlying cause



FOLLOW-UP CARE

PATIENT MONITORING

- Hydration status by clinical assessment of hydration and serial evaluation of body weight
- Fluid intake and urine output—provide a useful baseline for assessing adequacy of hydration therapy

POSSIBLE COMPLICATIONS

- Dehydration

EXPECTED COURSE AND PROGNOSIS

- Vary with underlying cause



KEY POINTS

- Polyuria—increased urination; greater than normal urine production
- Polydipsia—increased thirst; greater than normal water consumption
- Serious medical consequences for the patient are rare, if patient has free access to water and is willing and able to drink; until the mechanism of increased urination (polyuria) is understood, access to water should not be limited to avoid possible dehydration (any

limitation of access to water should be considered only under the direction of your pet's veterinarian)

CONGENITAL PORTOSYSTEMIC SHUNT OR VASCULAR ANOMALY



BASICS

OVERVIEW

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly—malformation of the veins connecting the portal and general body (systemic) circulations, permitting portal blood to bypass the liver; the portal vein is the vein that normally carries blood from the digestive organs to the liver
- Malformation of the veins may be within the liver (known as an “intrahepatic shunt”), more common in small-breed dogs and cats, or outside the liver (known as an “extrahepatic shunt”), more common in large-breed dogs
- Most shunts are single blood vessels
- May have other blood vessel (vascular) abnormalities involving the portal vein or small blood vessels within the liver (known as “intrahepatic microvasculature”)
- Acquired (condition that develops sometime later in life/after birth) portosystemic shunt (condition of abnormal blood flow in the liver due to high blood pressure in the portal vein [portal hypertension], the vein carrying blood from the digestive organs to the liver)—can develop following surgical “tying off” or “ligating” of the congenital (present at birth) abnormal blood vessel
- The liver is the largest gland in the body; it has many functions, including production of bile (a fluid substance involved in digestion of fats); production of albumin (a protein in the plasma of the blood); and detoxification of drugs and other chemicals (such as ammonia) in the body

GENETICS

- Genetically transmitted in high-risk breeds
- Affected breeds—Yorkshire terriers, Cairn terriers, Maltese, Tibetan spaniels, Havanese, Irish wolfhounds; Old English sheepdogs
- Suspect inheritance as a dominant trait, with incomplete penetrance

SIGNALMENT/DESCRIPTION OF ANIMAL

Species

- Dogs and cats

Breed Predilections

- Higher risk—purebred dogs; mixed-breed cats
- Especially common in the following dog breeds: Yorkshire terrier, Maltese, Cairn terrier, Tibetan spaniel, Havanese, Irish wolfhound

Mean Age and Range

- Usually first identified in juvenile animals; but dogs have been as old as 13 years of age at first diagnosis
- Accidental discovery of presence of portosystemic shunt: older animals that do not have clinical signs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Episodic brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”)—episodes transiently improve with fluid therapy, broad-spectrum antibiotics, and lactulose
- Cats initially thought to have upper respiratory infection (due to display of excessive drooling [known as “ptyalism”], which is a sign of hepatic encephalopathy in cats)
- Signs initiate with weaning of puppy or kitten to commercial food
- Animal may have normal appearance or a stunted stature; stunted growth—common
- Central nervous system signs—weakness; pacing; wobbly, incoordinated or “drunken” appearing gait (known as “ataxia”); disorientation; head pressing; blindness; behavioral changes: aggression (cats), vocalization, hallucinations; seizures; coma
- Gastrointestinal signs—lack of appetite; vomiting; diarrhea; eating of nonfood items (known as “pica”)
- Urinary signs—increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”); presence of ammonium biurate crystals in the urine; abnormal frequent passage of urine (known as “pollakiuria”); difficult or painful urination (known as “dysuria”); blood in the urine (known as “hematuria”); blockage or obstruction of the urethra (the tube from the bladder to the outside, through which urine flows out of the body) and rarely the ureters (the tubes from the kidneys to the bladder) due to the presence of ammonium biurate urinary tract stones (known as “ammonium biurate uroliths”)
- Some dogs lack clinical signs
- Affected female dogs (bitches) may produce litters

- Affected dogs may be used at stud before diagnosis recognized
- Small liver (known as “microhepatica”)
- Copper-colored irises in non–blue-eyed, non-Persian cats
- Fluid build-up in the abdomen (known as “ascites”) or in other tissues of the body (known as “edema”)—rare

CAUSES

- Congenital (present at birth) malformation of blood vessels
- Acquired (condition that develops sometime later in life/after birth) portosystemic shunt in animals with congenital (present at birth) portosystemic vascular anomaly may develop subsequent to increased blood pressure in the portal vein (portal hypertension), either congenital portal hypertension or surgically induced portal hypertension following surgical “tying off” or “ligating” of the abnormal blood vessel

RISK FACTORS

- Portosystemic shunt or vascular anomaly—purebred dogs, especially small terrier-type breeds
- The Irish wolfhound appears to have slow closure of the fetal blood vessel (known as the “ductus venosus”) that carries blood from the umbilical vein to the vena cava; the “vena cava” is the main vein that returns blood from the body to the heart



TREATMENT

HEALTH CARE

- Inpatient—severe signs of hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia); supportive care and initiation of medical management prior to liver biopsy and surgical ligation
- Hepatic encephalopathy should be treated medically before surgery to “tie off” or “ligate” blood vessels

DIET

- Nutritional support—essential to maintain body condition, as muscle serves as an important site of temporary ammonia detoxification
- Balanced, protein-restricted diet—recommended; thereafter, protein allocation based on response in combination with treatment for hepatic encephalopathy; as tolerated, add protein (use cottage cheese or calcium caseinate in dogs), as directed by your pet’s veterinarian

SURGERY

- Congenital (present at birth) portosystemic shunt or portosystemic vascular anomaly (in which blood flows abnormally between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)—surgical correction (in which the abnormal blood vessel is “tied off” or “ligated” using Ameroid constrictor or cellophane banding) is a possibility in many cases
- Surgical ligation—optimal goal is total ligation, but this may not be tolerated in some dogs
- Partial ligation only achieved in many dogs
- Ameroid constrictor—reduces immediate surgical risks of ligation; may later result in acquired (condition that develops sometime later in life/after birth) portosystemic shunt in some patients (especially Yorkshire Terriers)
- Portosystemic vascular anomaly within the liver (intrahepatic)—most difficult to ligate
- Emergency surgery—sometimes required for removal of ligature or Ameroid constrictor
- Fluid build-up in the abdomen (ascites)—common after shunt ligation; may be sign of increasing blood pressure in the portal vein (portal hypertension)
- Intensive care (ICU) monitoring—recommended postoperatively for 72 to 96 hours



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medical management is directed at treatment of hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)
- Medications that increase dietary protein tolerance, change bacteria or conditions in the intestines, reduce production or availability of

substances provoking hepatic encephalopathy

- Antibiotics—antibiotic selection based on ability to change the bacteria in the intestines or their products; administered by injection (known as “systemic administration”); antibiotics such as metronidazole or amoxicillin; combine use with lactulose
- Nonabsorbable-fermented carbohydrates—lactulose, lactitol, or lactose (if lactase deficient); decrease production or absorption of ammonia; increase rate of stool transit; trap nitrogen in bacteria; lactulose most commonly used; therapeutic goal is passage of two to three soft stools daily; also may be administered as an enema for sudden (acute) hepatic encephalopathy and coma *after* cleansing enemas have removed debris
- Enemas—*cleansing enemas* (warmed polyionic fluids) mechanically clean colon; *retention enemas* directly deliver fermentable substrates or directly alter colonic pH and organisms: diluted lactulose, lactitol, or lactose; neomycin in water; diluted Betadine®
- Zinc supplementation, as directed by your pet’s veterinarian
- Fluid build-up in the brain (known as “cerebral edema”)—complicates sudden (acute) hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia); administer medication (mannitol) to decrease fluid build-up; administer nasal oxygen and *N*-acetylcysteine; use of steroids to decrease fluid build-up (edema) is controversial as steroids may promote bleeding in the intestinal tract (which is a risk factor for development of hepatic encephalopathy)
- If epileptic seizure activity—zonisamide or potassium bromide is the preferred medication to control seizures (known as an “anticonvulsant”) compared to phenobarbital



FOLLOW-UP CARE

PATIENT MONITORING

- Following surgical ligation of blood vessels, watch closely for signs of lack of blood flow to the intestines (such as bloody diarrhea, abdominal pain, failure to recover from surgery/anesthesia, unexplained rapid heart rate [known as “tachycardia”], increased body temperature [known as “hyperthermia”] or decreased body temperature [known as “hypothermia”]); monitor girth and body weight
- Reevaluate patient’s at-home behavior; body condition; girth circumference; blood work (complete blood count [CBC] and serum biochemistry panel), and urinalysis (looking for resolution of ammonium biurate crystals in the urine [crystalluria])

PREVENTIONS AND AVOIDANCE

- If multiple abnormal blood vessels (portosystemic shunts) are identified, they likely are acquired (condition that develops sometime later in life/after birth) portosystemic shunts—do not pursue surgical ligation; another underlying liver disease or disorder is causing increased blood pressure in the portal vein (portal hypertension)

POSSIBLE COMPLICATIONS

- Postoperative complications—blood clots in the portal vein (known as “portal venous thrombi”); sudden (acute) severe high blood pressure in the portal vein (portal hypertension); lack of blood flow to the intestines; accumulation of bacterial toxins in the blood (known as “endotoxemia”); seizures; generalized bacterial infection (known as “sepsis”); sudden (acute) inflammation of the pancreas (known as “pancreatitis”); bleeding
- Low body temperature (hypothermia) during or following surgery—especially in very small patients; complicates recovery
- Seizures following surgical ligation

EXPECTED COURSE AND PROGNOSIS

- Cannot predict individual response to surgery
- Dogs—surgical ligation improves signs in 70% to 80% of patients with clinical signs of portosystemic shunt
- Cats—many develop acquired (condition that develops sometime later in life/after birth) portosystemic shunt with ligation
- Following surgery—continue management of hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia) until reevaluation of clinical status
- Some patients require indefinite treatment
- Increased risk of poor outcome in certain small dogs and cats
- Despite initial good response, recurrence of shunting may develop after 3 years
- Dogs with portosystemic vascular anomaly that do not have clinical signs and have not had surgery can lead a full life expectancy



KEY POINTS

- Surgical ligation—expect improvement but not cure; may not be required for all dogs, as some respond well to feeding a commercial

diet manufactured for patients with hepatic encephalopathy (brain disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)

- Clinical signs may persist despite surgical intervention (ligation), requiring long-term (chronic) nutritional and medical management

INFLAMMATION OF THE PROSTATE (PROSTATITIS) AND PROSTATIC ABSCESS



BASICS

OVERVIEW

- “Prostatitis” is inflammation of the prostate
- “Prostatic abscess” is an abscess (a localized accumulation of pus, leading to tissue damage and inflammation) of the prostate

Sudden (Acute) Prostatitis

- Infection of the canine prostate with bacteria, *Mycoplasma*, and/or fungi, with generalized (systemic) signs of fever; lack of appetite (anorexia); sluggishness (lethargy); pain; and inflammatory cells (white-blood cells) in prostatic fluid; presence of abscesses is variable, occurring in 15 of 25 dogs with inflammation of the prostate (prostatitis) in one study—abscesses occasionally rupture into the abdominal cavity, causing generalized bacterial infection (known as “sepsis”), shock, and rarely death

Long-Term (Chronic) Prostatitis

- Infection without detectable signs in the animal (known as “subclinical infection”) of the canine prostate, in the absence of prostatic abscesses and signs of generalized disease or multiple organ involvement; the infection may be recent or long-term; affected dogs do not have signs of disease, except for the presence of inflammatory cells (white-blood cells) in the prostatic fluid, which causes infertility
- Long-term (chronic) inflammation of the prostate (prostatitis) may occur after or independently of sudden (acute) prostatitis

GENETICS

- No known genetic basis

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilection

- All breeds and mixed-breed dogs

Mean Age and Range

- Middle-aged; mean age range, 7 to 11 years

Predominant Sex

- Intact male dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

Sudden (Acute) Prostatitis

- Lethargy/depression
- Lack of appetite (anorexia)
- Straining to defecate (known as “tenesmus”)
- Difficulty urinating (known as “dysuria”)
- Fever (known as “pyrexia”)
- Pain at prostatic or caudal abdominal palpation
- Bloody discharge from the urethra
- Stiff hind-limb gait
- Shock associated with generalized bacterial infection (sepsis; condition known as “septic shock”)—rare

Long-Term (Chronic) Prostatitis

- May have no detectable signs
- Straining to defecate (tenesmus)
- Difficulty urinating (dysuria)
- Bloody discharge from the urethra

CAUSES

- Infection of the enlarged prostate with bacteria moving up the urethra; possible bacteria include *Escherichia coli*, *Staphylococcus*, *Streptococcus*, *Proteus mirabilis*, *Klebsiella*, *Enterobacter*, *Hemophilus*, *Pseudomonas*, *Pasteurella*, anaerobic bacteria (bacteria that can

live and grow in the absence of oxygen), and *Mycoplasma* (most common)

- Infection of the enlarged prostate from a generalized (systemic) bacterial infection in the body, including *Brucella canis*
- Generalized (systemic) or local puncture wound infection with *Blastomyces dermatitidis* (a fungus)

RISK FACTORS

- Increasing age; incidence is high in intact male dogs over five years of age
- Presence of functional testicles in affected dogs
- Enlarged prostate (known as “benign prostatic hypertrophy” [BPH])
- Historical administration of male hormone (androgen) or female hormone (estrogen)
- Impaired host-defense mechanisms (such as decreased ability to develop an immune response [known as “immunosuppression”], catheterization of the urethra)



TREATMENT

HEALTH CARE

- Sudden (acute) inflammation of the prostate (prostatitis), prostatic abscess, and rupture of prostatic abscesses into the abdominal cavity are potentially life-threatening emergencies that can lead to shock associated with generalized bacterial infection (septic shock) and death; affected patients should be hospitalized and diagnostic samples (blood, urine, semen, imaging) collected immediately
- Dogs with sudden (acute) prostatitis or prostatic abscess should receive antibiotics administered through an intravenous line
- The patient should be assessed for likelihood of abscess rupture and inflammation of the lining of the abdomen (known as “peritonitis”), which warrants intravenous fluid therapy for shock associated with generalized bacterial infection (septic shock)
- Dogs with long-term (chronic) prostatitis may be seen as outpatients for diagnostic procedures, and started on specific therapy when laboratory results are available

ACTIVITY

- Breeding should be avoided until bacteria have been cleared from the prostatic fluid

SURGERY

- Surgical management of prostatic abscesses should be deferred until after initiation of antibiotics and treatment to return the enlarged prostate to a more normal size (known as “prostatic involution”); involution is associated with resolution of abscesses, often making surgery unnecessary
- Castration or neutering is recommended for induction of prostatic involution in non-breeding dogs with inflammation of the prostate (prostatitis); castration should be deferred until after identification and treatment (for at least one week) of the causative bacterial/fungal agent; alternatively, medical treatment to lead to involution of the prostate may be induced with finasteride
- A variety of surgical techniques have been suggested for treatment of prostatic abscesses in dogs; however, these procedures have been associated with a high percentage of short- and long-term undesirable consequences, including recurrence of abscesses



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Eradicating Infection

- Choice of antibiotics is based on bacterial and/or fungal culture and susceptibility findings in the prostatic fluid, antibiotic lipid solubility (which enhances its ability to move into prostatic tissue in therapeutic concentrations), and assessment of sudden (acute) or long-term (chronic) status of the infection
- Antibiotics of choice in long-term (chronic) inflammation of the prostate (prostatitis) are those known to move into normal prostatic tissue in therapeutic concentrations, including chloramphenicol, erythromycin, fluoroquinolones, and trimethoprim; in sudden (acute) prostatitis, the blood-prostate barrier is disrupted, and almost any antibiotic will reach the prostatic tissue in therapeutic concentrations
- Emergency antibiotic treatment of choice in dogs with sudden (acute) prostatitis and/or abscess, administered after collection of prostatic fluid for culture, is amoxicillin/clavulanate with enrofloxacin

Inducing Prostatic Involution (treatment to return the enlarged prostate to a more normal size)

- Treatment of choice for inducing permanent prostatic involution is castration

- Alternatively, finasteride for 2 to 4 months induces involution of the prostatic tissue
- Finasteride prevents conversion of testosterone to dihydrotestosterone (DHT; the hormone that is primarily involved in development of the prostate), thereby causing prostatic involution without adversely affecting libido or sperm production
- Enlargement of the prostate (benign prostatic hypertrophy or BPH) recurs following cessation of finasteride therapy



FOLLOW-UP CARE

PATIENT MONITORING

- Repeated evaluation of semen culture, cytology, and prostatic imaging
- Intervals between re-evaluations vary with severity of signs, presence of an abscess, selection of castration or finasteride therapy for prostatic involution, and use of the dog in a breeding program; intervals between evaluations range from 1 to 8 weeks, with recheck recommended prior to breeding
- Continue patient monitoring until the dog has been castrated

PREVENTIONS AND AVOIDANCE

- Castration is recommended to induce prostatic involution (to return the enlarged prostate to a more normal size), resolution of benign prostatic hypertrophy (BPH), and prevention of recurrence

POSSIBLE COMPLICATIONS

- Recurrence of infection, if prostatic involution (to return the enlarged prostate to a more normal size) is not induced
- Surgical drainage of abscesses is associated with many complications, including lack of control of urination (known as “urinary incontinence”); recurrence of abscesses; low levels of protein in the blood (known as “hypoproteinemia”); fluid build-up in the scrotum (known as “scrotal edema”); low red-blood cell counts (known as “anemia”); generalized bacterial infection (sepsis); and shock

EXPECTED COURSE AND PROGNOSIS

- Prognosis is good to excellent, except in the case of rupture of prostatic abscesses into the abdominal cavity, with resulting inflammation of the lining of the abdomen (peritonitis)
- Castration prevents recurrence and improves prognosis
- Surgical management of prostatic abscesses is associated with complications and a poorer prognosis than medical/surgical induction of prostatic involution (to return the enlarged prostate to a more normal size)



KEY POINTS

- Castration should be recommended for dogs with sudden (acute) inflammation of the prostate (prostatitis) and/or prostatic abscess, as castration induces permanent prostatic involution (to return the enlarged prostate to a more normal size)
- If maintenance of breeding potential is necessary, long-term or intermittent treatment with finasteride is recommended to induce prostatic involution; routine rechecks at 2 to 3 month intervals for semen culture, semen cytology, and prostatic imaging are recommended
- Prostatic enlargement (benign prostatic hypertrophy or BPH) recurs over time in intact male dogs after treatment with finasteride is discontinued, and BPH increases risk of recurrence of inflammation of the prostate (prostatitis)

PROTEINURIA (PROTEIN IN THE URINE)



BASICS

OVERVIEW

- “Proteinuria” is the medical term for protein in the urine
- Proteinuria is a subjective increase in urinary protein as detected by urine dipstick analysis, urinary protein: creatinine ratio (UP:C of 0.5 or higher) or a 24-hour urine protein content over 20 mg/kg body weight
- “Microalbuminuria” is a type of proteinuria in which a low, yet abnormal, concentration of albumin (a type of protein) is present in the urine
- Proteinuria can be caused by changes or diseases involving the body, changes involving the kidney itself, or changes after the urine leaves the kidneys (such as inflammation of the bladder [known as “cystitis”])
- The kidney filters the blood and removes various waste products from the body as it produces urine; the kidney is involved in maintaining the normal fluid volume of the body; each kidney is composed of thousands of nephrons (the functional units of the kidney, each consisting of the glomerulus [a tuft of blood capillaries—the “blood filter”] and a series of tubes and ducts, through which the filtered fluid flows, as urine is produced)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats (less common)

Breed Predispositions

- Protein in the urine originating from the glomerulus of the kidney (known as “glomerular proteinuria”) may be the initial manifestation of familial (runs in certain families or lines of animals) kidney disease in soft-coated wheaten terriers, bull terriers, English cocker spaniels, Dalmatians, Samoyeds, beagles, Bernese mountain dogs, and Chinese shar peis

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vary with underlying cause and severity of protein in the urine (proteinuria)
- None directly attributed to the presence of protein in the urine (proteinuria)

CAUSES

Prerenal Proteinuria (protein in the urine originates from changes or disease in the body, before the kidney)

- Functional proteinuria—strenuous exercise, fever, low body temperature (known as “hypothermia”), seizures, or accumulation of an increased volume of blood in the veins (known as “venous congestion”); poorly documented as a cause of proteinuria in dogs and cats
- Overload proteinuria—large amounts of low molecular-weight plasma proteins in the glomerular filtrate (such as from excessive breakdown of red-blood cells [known as “hemolysis”] or destruction of skeletal muscles [known as “rhabdomyolysis”], production of abnormal proteins or Bence-Jones proteins secondary to cancer) overwhelms the ability of the kidneys to resorb the protein

Renal Proteinuria (protein in the urine originates from changes or disease in the kidney itself)

- “Glomerulonephritis” is inflammation and accompanying dysfunction of glomeruli (plural of glomerulus) of the kidney; inflammation most commonly is due to the presence of immune complexes in the glomerulus; examples are membranoproliferative glomerulonephritis and proliferative glomerulonephritis
- Glomerular disease of any type (known as “glomerulonephropathy,” such as membranous nephropathy)
- Inherited inflammation of the kidney (known as “hereditary nephritis”)
- A group of conditions of differing cause in which insoluble proteins (amyloid) are deposited outside cells in the kidneys and various other organs, compromising the normal function of the kidney and other organs (condition known as “amyloidosis”)
- Scar tissue involving the blood vessels (capillaries) of the glomerulus (known as “glomerulosclerosis”); scar tissue develops following some type of injury to the glomerulus
- In general, amyloidosis results in the heaviest amount of protein in the urine (proteinuria), although dogs with other glomerular diseases also can have very heavy proteinuria
- Tubular dysfunction resulting in failure of tubular protein reabsorption can cause mild-to-moderate proteinuria

Postrenal Proteinuria (protein in the urine originates from changes or disease after the urine leaves the kidneys)

- Bleeding or inflammation of the urinary bladder, urethra (the tube from the bladder to the outside, through which urine flows out of the body), and reproductive organs (such as the vagina, penis, prostate)

RISK FACTORS

- Long-term (chronic) inflammatory disease (such as infectious and immune-mediated disease) and cancer can lead to development of

glomerulonephritis (inflammation of the glomerulus, usually due to the presence of immune complexes) or amyloidosis (condition in which insoluble proteins [amyloid] are deposited outside cells in the kidneys)

- High blood pressure (known as “hypertension”)
- Long-term (chronic) high levels of lipid (a group of compounds that contain fats or oils) in the blood (known as “hyperlipidemia”); for example, as seen in miniature schnauzers
- Blood in the urine (known as “hematuria”) and pus in the urine (known as “pyuria”)
- Multiple myelomas can produce Bence-Jones proteins, which may be found in the urine



TREATMENT

HEALTH CARE

- Most dogs and cats with protein in their urine (proteinuria) can be managed as outpatients
- Inpatient care may be required during diagnostic evaluation (such as kidney biopsy) or when complications are associated with kidney failure

ACTIVITY

- Maintain normal activity if protein in the urine (proteinuria) is the only laboratory abnormality
- Physical therapy and exercise may limit formation or assist in the mobilization of fluid build-up (known as “edema”) in patients with protein in their urine from the glomeruli of the kidney (glomerular proteinuria) and low levels of albumin in their blood (known as “hypoalbuminemia”)—cage confinement should be avoided for these patients

DIET

- If glomerular disease is suspected, feed a diet moderately reduced in protein, reduced in sodium and rich in omega-3 fatty acids
- Diets formulated for kidney failure are appropriate

SURGERY

- Kidney biopsy is needed to diagnose the glomerular disease specifically, when an underlying disease cannot be identified or protein in the urine (proteinuria) has persisted for several months following treatment of the underlying disease



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Angiotensin-converting enzyme (ACE) inhibitors should be given to dogs, and possibly cats, with protein in their urine from the glomeruli of the kidney (glomerular proteinuria)
- The use of aldosterone antagonists in the management of protein in the urine (proteinuria) is being investigated
- Antibiotics may be indicated, depending on the underlying cause of the protein in the urine (proteinuria)



FOLLOW-UP CARE

PATIENT MONITORING

- The urine protein:creatinine ratio (UP:C) or urine albumin content should be used to assess progression of glomerular disease and response to treatment and should be evaluated for months after resolution of any treatable underlying disease
- Monitor serum creatinine; reduced levels of protein in the urine (proteinuria) or reduced levels of albumin (a protein) in the urine (known as “albuminuria”) may reflect deteriorating kidney function

PREVENTIONS AND AVOIDANCE

- All adult dogs and cats should have annual urinalyses, which should include determination of urine protein and/or albumin; if protein in

the urine (proteinuria) or albumin (a protein) in the urine (albuminuria) is detected, the tests should be repeated in 2 to 4 weeks

- Dogs or cats with persistent protein in the urine (proteinuria) or low levels of albumin in the urine (microalbuminuria) of glomerular origin should be evaluated more thoroughly for underlying causes of glomerular injury

POSSIBLE COMPLICATIONS

- Fluid build-up (edema)
- Blood clots (known as “thromboembolism”)
- High blood pressure (hypertension)
- Progressive kidney disease
- Poor wound healing

EXPECTED COURSE AND PROGNOSIS

- Vary with the cause of protein in the urine (proteinuria)
- Postrenal and prerenal proteinuria should resolve following resolution of the inciting cause
- Most diseases associated with renal tubular proteinuria will be progressive
- Although glomerular diseases often are progressive, the rate of progression varies and spontaneous remissions have been reported
- Animals with persistent protein in their urine from the glomeruli of the kidney (glomerular proteinuria) may develop kidney tubular damage, resulting in kidney failure and eventual death
- Some dogs die shortly after the initial detection of protein in the urine (proteinuria), while others remain alive for years



KEY POINTS

- Kidney biopsy is needed to diagnose the glomerular disease specifically, when an underlying disease cannot be identified or protein in the urine (proteinuria) has persisted for several months following treatment of the underlying disease
- Some dogs die shortly after the initial detection of protein in the urine (proteinuria), while others remain alive for years

ITCHINESS (PRURITUS)



BASICS

OVERVIEW

- “Pruritus” is the medical term for itching or itchiness; it is the itching sensation that provokes the desire to scratch, rub, chew or lick
- Pruritus is an indicator of inflamed skin
- The term is not a diagnosis, but rather is a description of a clinical sign

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Scratching
- Licking
- Biting
- Chewing
- Self-trauma
- Inflammation of the skin (known as “dermatitis”)
- Hair loss (known as “alopecia”)
- Other signs determined by underlying cause

CAUSES

- Parasites—fleas; mites (canine scabies [*Sarcoptes*], *Demodex*, ear mites [*Otodectes*], feline scabies [*Notoedres*], “walking dandruff” [*Cheyletiella*], harvest mite or red bud [*Trombicula*]); lice; rhabditic dermatitis (*Pelodera strongyloides*); or migration of internal parasites
- Allergies—parasite allergy; atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems); food allergy; contact allergy; drug allergy; allergy to skin bacteria (known as “bacterial hypersensitivity”)
- Bacterial or fungal infections—typically *Staphylococcus* (a bacteria) and *Malassezia pachydermatis* (a yeast or fungus); rarely a dermatophyte (fungus living on the skin, hair, or nails); however, *Trichophyton* is a dermatophyte that tends to cause more itchy skin disease than the other dermatophytes
- Miscellaneous—excessive scaling of the skin (known as “seborrhea”); calcium deposits in the skin (known as “calcinosis cutis”); skin tumors or cancer
- Immune-mediated skin diseases and hormonal skin diseases can be variably itchy
- Psychological skin diseases may be associated with itchiness

RISK FACTORS

- Exposure to other animals with parasites



TREATMENT

HEALTH CARE

- More than one disease can contribute to itching
- If identification and treatment for one cause of itchiness does not result in adequate improvement, consider other coexistent causes
- The use of mechanical restraint (such as an Elizabethan collar) can be a helpful option, but is seldom feasible in long-term treatment

DIET

- Depends on underlying cause

- Usually no change in diet needed, unless suspect food allergy

SURGERY

- Skin biopsy may be necessary for diagnosis or to determine underlying cause



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Medications Applied to the Skin Directly (Known as “Topical Therapy”)

- Topical therapy is helpful in mildly itchy pets
- For localized areas of itchiness or skin inflammation, sprays, lotions and creams are most appropriate
- If the itching involves many areas or widespread areas, shampoos are the preferred means of application
- Antibacterial shampoos help control bacterial infections that cause itching; however, some antibacterial shampoos (such as those containing benzoyl peroxide or iodine) can cause increased itching
- Colloidal oatmeal can be found in virtually all forms of topical therapy; in some cases, it is very beneficial, but its duration of effect usually is less than two days
- Topical antihistamines may be found alone or in combination with other ingredients; they may not have a beneficial effect
- Topical anesthetics may offer only a very short duration of effect
- Lime sulfur (which has a bad odor and can stain) can decrease itching, while also having anti-parasitic, antibacterial, and antifungal properties
- Topical steroids probably are the most useful topical medications; hydrocortisone is the mildest and most common topical steroid; stronger steroids (such as betamethasone) may be more effective and are more expensive
- Some topical steroid medications also contain ingredients (such as alcohol), which can aggravate already irritated skin
- In some animals, the application of any substance, including water (especially warm water), can result in an increased level of itchiness; however, cool water often is soothing

Medications Administered by Mouth or by Injection (Systemic Therapy)

- Steroids to decrease inflammation and itchiness of the skin
- Cyclosporine to decrease the immune response
- For patients affected with airborne allergies for more than a few months out of the year, “allergy shots” (known as “allergen specific immunotherapy”) are appropriate, frequently beneficial, and may lead to a cure (in some cases)
- Antihistamines (such as hydroxyzine, diphenhydramine, and chlorpheniramine) to prevent inflammation and itching
- Fatty acids are available in powder, liquid, and capsules; they help block pathways that lead to inflammation, but may require 6 to 8 weeks of use until maximum effect is observed; fatty acids work better as preventive medications, rather than stopping the inflammation once it has become a problem; they also help control dry or flaky skin, which can cause itching
- Medications to relieve anxiety or depression (known as “psychogenic drugs”) can be helpful in controlling itchiness; include such drugs as amitriptyline, fluoxetine, and diazepam
- The use of drugs other than steroids to control itching is less convenient, but reduces potential for serious side effects—if these other drugs are not totally effective in controlling clinical signs, they often help reduce the amount of steroids that is necessary to decrease itchiness
- In extremely rare cases, medications to decrease the immune response (known as “immunosuppressive drugs,” such as azathioprine) may be utilized; however, they generally are reserved for instances where euthanasia is being considered or because all other treatment has failed



FOLLOW-UP CARE

PATIENT MONITORING

- Patient monitoring is imperative; pets should be examined periodically to evaluate response to treatment
- Patients receiving long-term (chronic) steroids should be evaluated every 3 to 6 months for signs of medication-induced excessive levels of steroids in the body (known as “iatrogenic Cushing’s disease”)

PREVENTIONS AND AVOIDANCE

- Prevent infestation with parasites (such as fleas and mites)
- Avoid foods identified as causing food allergy for your pet

POSSIBLE COMPLICATIONS

- Owner frustration is common
- Complications (such as increased thirst [known as “polydipsia”] and increased urination [known as “polyuria”]) are common with long-term (chronic) steroid use

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause
- Many causes of itchiness in pets are extremely frustrating to control



KEY POINTS

- Many different unrelated diseases may contribute to itchiness (pruritus) and control of one disease does not mean that other causes cannot be contributing to itchiness or cannot occur later
- Multiple causes (such as flea allergy, inhalant allergy, and bacterial skin infection [known as “pyoderma”]) commonly are present in a single patient
- Elimination of bacterial skin infection (pyoderma) and flea-associated disease may not be enough to significantly reduce itchiness
- Food-allergy and inhalant-allergic animals may do well during the winter season with a hypoallergenic diet, only to become itchy during the warmer months in association with inhalant allergies

PULMONIC STENOSIS



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- “Pulmonic” refers to pulmonary; “pulmonary” refers to the lung(s); “pulmonic stenosis” is a condition characterized by narrowing of the pulmonary valve in the heart, which is the valve from the right ventricle to the main pulmonary artery
- “Stenosis” is the medical term for narrowing
- “Pulmonic stenosis” is a congenital (present at birth) narrowing at some point in the area through which blood flows out of the right ventricle, through the pulmonary valve, and into the main pulmonary artery (main artery of the lungs); this area is known as the “right ventricular outflow tract;” the narrowing blocks or obstructs the flow of blood from the right ventricle to the pulmonary artery
- Defect can be at the valve itself (known as “valvular pulmonic stenosis”), below the valve (known as “subvalvular pulmonic stenosis,” or above the valve, just inside the pulmonary artery (known as “supravalvular pulmonic stenosis”); valvular pulmonic stenosis is the most common form

GENETICS

- Inherited defect in beagles; multiple genes probably are involved (known as a “polygenic mode of transmission”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- English bulldog, Scottish terrier, wirehair fox terrier, miniature schnauzer, West Highland white terrier, Chihuahua, Samoyed, mastiff, cocker spaniel, beagle, boxer

Mean Age and Range

- Present from birth and may be detected as a heart murmur in puppies
- If a heart murmur is not detected, affected animals may not be identified until clinical signs develop later in life

Predominant Sex

- Males are more likely than females to have pulmonic stenosis in English bulldogs and possibly other breeds

SIGNS/OBSERVED CHANGES in the ANIMAL

- Mild narrowing (stenosis)—usually no clinical signs
- Severely affected patients—may develop congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs), fainting with exertion (known as “exertional syncope”), or sudden death
- Abdominal swelling or distension
- Difficulty breathing (known as “dyspnea”)
- Exercise intolerance
- Heart murmur
- May be able to feel vibrations caused by abnormal blood flow (known as “thrills”) when placing hand against the chest wall (generally associated with more severe narrowing [stenosis])
- Irregular heart beats (known as “arrhythmias”)
- Rapid heart rate (known as “tachycardia”) may occur if pet is in congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)
- Other signs of congestive heart failure include fluid build-up in the abdomen (known as “ascites”), enlargement of the jugular veins in the neck, and rapid breathing (known as “tachypnea”)

CAUSES

- Congenital (present at birth) heart defect



TREATMENT

HEALTH CARE

- Most managed as outpatients
- Initial hospitalization of those pets with severe congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Rarely, fluid build-up in the space between the lungs and chest wall (known as “pleural effusion”) may need draining; fluid build-up in the abdomen (ascites) usually is treated medically

ACTIVITY

- Exercise should be restricted in pets with fainting (syncope) or congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs)
- Excessive exertion should be avoided in pets with severe narrowing (stenosis) that do not have clinical signs

DIET

- Low-salt diets may benefit those pets with fluid build-up in the abdomen that does not respond to medical treatment (known as “refractory ascites”)

SURGERY

- Balloon dilation (procedure in which an instrument with an expandable balloon is inserted into the area of the narrowing [stenosis] and the balloon is expanded to open the narrowing) of the outflow tract, performed during heart catheterization
- Alternative surgical techniques include cutting through the narrowed pulmonary valve to relieve blockage to blood flow (procedure known as a “valvulotomy”) or patch-graft procedures; mortality rates tend to be higher than with balloon dilation



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- If pet has signs of congestive heart failure, treat fluid build-up in the abdomen (ascites) with medications to remove excess fluids from the body (known as “diuretics”), such as furosemide or spironolactone (used as an additional diuretic in cases that do not respond to medical treatment); treat atrial fibrillation (rapid, irregular heart rhythm involving the top two chambers of the heart [atria]) with digoxin
- Medications to enlarge or dilate blood vessels (known as “vasodilators,” such as hydralazine) may cause low blood pressure (known as “hypotension”) without relieving the narrowing (stenosis) and are best avoided



FOLLOW-UP CARE

PATIENT MONITORING

- Use serial follow-up echocardiograms (use of ultrasound to evaluate the heart and major blood vessels)

PREVENTIONS AND AVOIDANCE

- Do not breed affected animals

POSSIBLE COMPLICATIONS

- Right-sided congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Irregular heart beats (arrhythmias)
- Exercise intolerance
- Fainting with exertion (exertional syncope)

- Sudden death

EXPECTED COURSE AND PROGNOSIS

- Mildly affected animals may remain without clinical signs and have normal life spans
- Severely affected animals have a guarded prognosis because they may develop congestive heart failure (condition in which the heart cannot pump an adequate volume of blood to meet the body's needs) or sudden death
- Clinical signs generally are more common in animals over 1 year of age



KEY POINTS

- Mildly affected animals may lead normal lives
- Moderately to severely affected patients may benefit from treatment interventions (such as balloon catheter dilation or surgery); improved clinical signs and survival have been associated with successful balloon procedures
- Prognosis is guarded once signs of congestive heart failure develop; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs
- Do not breed affected animals.

BACTERIAL INFECTION OF THE KIDNEY (PYELONEPHRITIS)



BASICS

OVERVIEW

- Bacterial infection/inflammation of the kidney (known as “pyelonephritis”)
- More descriptive term is “upper urinary tract infection” as the bacterial infection/inflammation may involve the kidney and urine collecting mechanism, as well as the ureters (the tubes from the kidney to the bladder)
- The upper urinary tract includes the kidneys and ureters; the lower urinary tract includes the bladder and urethra (the tube from the bladder to the outside, through which urine flows out of the body)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Dogs affected more commonly than cats

Mean Age and Range

- Mean age of affected dogs and cats unknown
- Dogs of any age can be affected
- Cats over 10 years of age are more likely to develop urinary tract infection than cats less than 10 years of age

Predominant Sex

- Unknown
- Dogs—urinary tract infection affects more females than males
- Cats—urinary tract infection is uncommon and occurs with similar frequency in males and females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Many pets have no signs or have signs of lower urinary tract infection only
- Increased urination (known as “polyuria” or “PU”) and increased thirst (known as “polydipsia” or “PD”)
- Abdominal or lumbar pain
- Pain upon palpation of kidneys
- Fever
- Signs associated with lower urinary tract infection—such as difficult or painful urination (known as “dysuria”); frequent voiding of small volumes of urine (known as “pollakiuria”); straining with slow, painful discharge of urine (known as “stranguria”); blood in the urine (known as “hematuria”); and malodorous or discolored urine

CAUSES

- Usually, bacterial infection moving up the urinary tract from a lower urinary tract infection; most common bacteria are *Escherichia coli* and *Staphylococcus*; other bacteria, including *Proteus*, *Streptococcus*, *Klebsiella*, *Enterobacter*, and *Pseudomonas*, which frequently infect the lower urinary tract, may move up into the upper urinary tract.
- Anaerobic bacteria (bacteria that can live and grow in the absence of oxygen), ureaplasma, and fungi rarely infect the upper urinary tract

RISK FACTORS

- Condition that occurs during fetal development, in which the ureters (the tubes from the kidneys to the bladder) do not attach to the bladder properly or may attach to reproductive organs instead of the bladder (known as “ectopic ureters”); reflux of urine from the bladder back into the ureters (known as “vesicoureteral reflux”); congenital (present at birth) abnormal development of the kidneys (known as “renal dysplasia”); and lower urinary tract infection
- Conditions that increase the likelihood of urinary tract infection—such as diabetes mellitus (“sugar diabetes”); increased levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); administration of medications containing steroids; kidney failure; catheterization of the urethra (the tube from the bladder to the outside, through which urine flows out of the body); urine retention; urinary tract stones (known as “uroliths”); urinary tract tumors; surgical removal of the penis with creation of a new opening into the urethra (known as “perineal urethrostomy”)
- In cats with lower urinary tract disease, indwelling urinary catheters combined with administration of steroids frequently results in bacterial infection/inflammation of the kidney (pyelonephritis)



TREATMENT

HEALTH CARE

- Outpatient, unless animal has generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”) or kidney failure

ACTIVITY

- Unlimited

DIET

- Modification recommended in animals with coexistent long-term (chronic) kidney failure or the presence of urinary tract stones in the kidney (known as “nephrolithiasis”)

SURGERY

- Surgically correct abnormally located ureters (ectopic ureters); the “ureters” are the tubes from the kidneys to the bladder
- Complete blockage or obstruction of the upper urinary tract by a urinary tract stone (urolith) in a patient with bacterial infection/inflammation of the kidneys (pyelonephritis) may result in generalized bacterial infection secondary to the urinary tract infection (known as “urosepsis”) and should be corrected by surgery or by a medical procedures in which the stone is broken up within the urinary tract using some type of energy or sound wave (procedures known as “lithotripsy”) for cases with kidney stones (known as “nephroliths”)
- Infected kidney stones (nephroliths)—surgically remove, medically dissolve (for struvite kidney stones), or fragment by extracorporeal shock wave lithotripsy a medical procedures in which the stone is broken up within the urinary tract using some type of energy or sound wave; use antibiotics at the time of the surgery or lithotripsy to reduce the risk of spreading bacterial infection into the body (urosepsis) when manipulating infected kidney stones (nephroliths)
- Surgical removal of one kidney (known as “unilateral nephrectomy”) usually is not effective for elimination of suspected bacterial infection/inflammation involving only that kidney (known as “unilateral pyelonephritis”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Base antibiotic selection on bacterial culture and sensitivity testing of the urine
- Antibiotics should kill bacteria (known as “bactericidal antibiotics”), achieve good serum and urine concentrations, and not be toxic to the kidneys
- High serum and urinary antibiotic concentrations do not necessarily ensure high tissue concentrations in certain areas of the kidney (known as the “renal medulla”); thus, long-term (chronic) bacterial infection/inflammation of the kidney (pyelonephritis) may be difficult to eradicate
- Give antibiotics by mouth for 4 to 6 weeks, as instructed by your pet’s veterinarian



FOLLOW-UP CARE

PATIENT MONITORING

- Do urine cultures and urinalyses during antibiotic administration (approximately 5 to 7 days into treatment) and 1 and 4 weeks after antibiotics are finished

PREVENTIONS AND AVOIDANCE

- Eliminate factors that increase the likelihood of urinary tract infection
- Surgically correct abnormally located ureters (ectopic ureters)

POSSIBLE COMPLICATIONS

- Kidney failure; recurrent bacterial infection/inflammation of the kidney (pyelonephritis); struvite urinary tract stones in the kidneys (known as “struvite nephroliths”); generalized disease caused by the spread of bacteria in the blood (septicemia or blood poisoning); shock associated with generalized bacterial infection (known as “septic shock”); and spread of infection to other areas of the body, such as infection/inflammation of the heart (known as “endocarditis”) and infection/inflammation of several joints (known as “polyarthritis”)

EXPECTED COURSE AND PROGNOSIS

- Patients with bacterial infection/inflammation of the kidney (pyelonephritis)—fair to good, with a return to normal health unless the patient also has kidney stones (nephroliths), long-term (chronic) kidney failure, or some other underlying cause for urinary tract infection (such as urinary tract blockage or obstruction or cancer)
- Established infection of the renal medulla may be difficult to resolve because of poor tissue penetration of antibiotics to this area of the kidney
- Patients with long-term (chronic) kidney failure caused by bacterial infection/inflammation of the kidney (pyelonephritis)—prognosis determined by the severity and rate of progression of the chronic kidney failure
- Recurrent bacterial infection/inflammation of the kidney (pyelonephritis) is likely, if infected kidney stones (nephroliths) are not removed



KEY POINTS

- Recurrent bacterial infection/inflammation of the kidney (pyelonephritis) may not cause clinical signs
- Unresolved long-term (chronic) bacterial infection/inflammation of the kidney (pyelonephritis) may lead to long-term (chronic) kidney failure; diagnostic follow-up is important to document resolution of pyelonephritis
- In patients with kidney stones (nephroliths), resolution of bacterial infection/inflammation of the kidney (pyelonephritis) is unlikely unless the kidney stones are removed

BACTERIAL INFECTION OF THE SKIN (PYODERMA)



BASICS

OVERVIEW

- “Pyoderma” is the medical term for bacterial infection of the skin
- Skin lesions are characterized by the presence of pus
- Bacterial infection of the skin (pyoderma) can be located in the top surface of the skin (known as the “epidermis”) and the hair follicles, in which case it is called a “superficial pyoderma” or can be located in the lower layers of the skin (known as the “dermis”), causing nodular abscesses in the skin (condition known as “furunculosis”) and draining tracts, in which case it is called a “deep pyoderma”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Dogs—very common
- Cats—uncommon

Breed Predilections

- Breeds with short coats, skin folds, or pressure calluses
- German shepherd dogs develop a severe, deep bacterial skin infection (pyoderma) that only partially may respond to antibiotics and frequently relapses

Mean Age and Range

- Age of onset usually related to underlying cause

SIGNS/OBSERVED CHANGES in the ANIMAL

- Superficial pyoderma (infection located in the top surface of the skin)—usually involves the trunk; extent of lesions may be hidden by the hair coat
- Deep pyoderma (infection located in the lower layers of the skin, causing nodular abscesses and draining tracts)—often affects the chin, bridge of the nose, pressure points, and feet; may be generalized
- Sudden (acute) or gradual onset
- Variable itchiness (known as “pruritus”)—underlying cause of the bacterial skin infection (pyoderma) may cause itchiness or the staphylococcal infection itself may cause itchiness
- Small, raised skin lesions (known as “papules”)
- Small, raised skin lesions containing pus (known as “pustules”)
- Large blood-filled sacs or blisters (known as “hemorrhagic bullae”)
- Dried discharge on the surface of the skin lesions (known as “crusts”)
- Circular patterns of hair loss (known as “alopecia”) bordered by scales (accumulations of surface skin cells, such as seen in dandruff) or surface peeling of the skin (the pattern is known as “epidermal collarettes”)
- Circular areas of reddened skin (known as “erythema”) or darkly pigmented skin (known as “hyperpigmentation”)
- Target lesions
- Loss of hair (alopecia), moth-eaten hair coat
- Accumulations of surface skin cells, such as seen in dandruff (known as “scales”)
- Thickening and hardening of the skin, usually associated with hyperpigmentation (known as “lichenification”)
- Abscess
- Nodular abscesses in the skin (furunculosis)
- Inflammation of the skin characterized by redness, swelling, and tenderness (known as “cellulitis”)

CAUSES

- *Staphylococcus intermedius*—most frequent
- *Pasteurella multocida*—an important disease-causing bacteria in cats
- Deep pyoderma (infection located in the lower layers of the skin, causing nodular abscesses and draining tracts)—may be complicated by gram-negative organisms (such as *E. coli*, *Proteus*, *Pseudomonas*)
- Rarely caused by higher bacteria (such as *Actinomyces*, *Nocardia*, *Mycobacteria*, *Actinobacillus*)

RISK FACTORS

- Allergy—flea-bite allergy; atopy (disease in which the animal is sensitized [or “allergic”] to substances found in the environment [such as pollen] that normally would not cause any health problems); food allergy; contact allergy (allergic reaction that develops following contact between the skin and the offending substance to which the animal is sensitized)
- Parasites—especially *Demodex* (cause of demodectic mange)
- Fungal infection—dermatophyte (a fungus living on the skin, hair, or nails)
- Hormonal disease—inadequate production of thyroid hormone (known as “hypothyroidism”); excessive production of steroids by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); sex-hormone imbalance
- Decreased ability to develop a normal immune response (known as “immune incompetency”)—steroids; young animals
- Excessively oily or dry scaling of the skin (known as “seborrhea”)—acne; condition seen in miniature schnauzers in which the hair follicles become filled with oil and skin cells that become infected (known as “schnauzer comedo syndrome”)
- Conformation—short coat; skin folds
- Trauma—pressure points; grooming; scratching; rooting behavior; irritants
- Foreign body—foxtail; grass awn



TREATMENT

HEALTH CARE

- Usually outpatient, except for severe, generalized deep bacterial infections of the skin (pyodermas, in which infection is located in the lower layers of the skin, causing nodular abscesses and draining tracts)
- Severe, generalized, deep bacterial infections of the skin (pyodermas)—may require intravenous fluids, antibiotics administered by injection, and daily whirlpool baths
- Benzoyl peroxide or chlorhexidine shampoos—remove surface debris
- Whirlpool baths—deep bacterial infections of the skin (pyodermas); remove crusted discharge; encourage drainage of pus

ACTIVITY

- No restriction

DIET

- Hypoallergenic diet, if bacterial infection of the skin (pyoderma) is secondary to food allergy; otherwise a high-quality, well-balanced food
- Avoid high-protein, poor-quality “bargain” diets and excessive supplementation

SURGERY

- Skin-fold bacterial infections of the skin (pyodermas) require surgical correction to prevent recurrence



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- *Staphylococcus intermedius* bacterial isolates—usually susceptible to cephalosporins, cloxacillin, oxacillin, methicillin, amoxicillin-clavulanate, erythromycin, and chloramphenicol; somewhat less responsive to lincomycin and trimethoprim-sulfonamide; frequently resistant to amoxicillin, ampicillin, penicillin, tetracycline, and sulfonamides
- Amoxicillin-clavulanate (type of antibiotic)—most isolates of *Staphylococcus* and *Pasteurella multocida* are susceptible; generally effective for skin infections in cats
- Superficial pyoderma (infection located in the top surface of the skin)—initially may be treated with one of the previously listed antibiotics, with antibiotic selection based on the veterinarian’s experience treating pyoderma (rather than based on results of bacterial culture and sensitivity testing)
- Recurrent, resistant, or deep pyoderma (infection located in the lower layers of the skin, causing nodular abscesses and draining tracts)—base antibiotic therapy on culture and sensitivity testing

- Multiple organisms with different antibiotic sensitivities—choose antibiotic on basis of staphylococcal susceptibility
- Vaccines containing *Staphylococcus* have been used in dogs with recurrent skin infections or with infections that respond poorly to treatment; examples include Staphage Lysate SPL®, Staphoid A-B, or autogenous bacterins—may improve antibiotic efficacy and decrease recurrence in a small percentage of cases



FOLLOW-UP CARE

PATIENT MONITORING

- Administer antibiotics for a minimum of 2 weeks beyond clinical cure; this is usually about 1 month for superficial pyodermas (infection located in the top surface of the skin), and 2 to 3+ months for deep pyodermas (infection located in the lower layers of the skin, causing nodular abscesses and draining tracts)

PREVENTIONS AND AVOIDANCE

- Routine bathing with benzoyl peroxide or chlorhexidine shampoos—may help prevent recurrences
- Some cases that continue to relapse may be managed with long-term/low-dose antibiotics, as directed by your pet's veterinarian
- Padded bedding—may ease pressure point-related bacterial infection of the skin (“pressure-point pyodermas”)
- Topical (applied to the skin directly) benzoyl peroxide gel or mupirocin ointment may be helpful additional therapies

POSSIBLE COMPLICATIONS

- Presence of bacteria in the blood (known as “bacteremia”) and generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”)

EXPECTED COURSE AND PROGNOSIS

- Likely to recur or not be responsive to medical treatment, if underlying cause is not identified and managed effectively



KEY POINTS

- “Pyoderma” is the medical term for bacterial infection of the skin
- Skin lesions are characterized by the presence of pus
- Bacterial infection of the skin (pyoderma) can be located in the top surface of the skin (epidermis) and the hair follicles, in which case it is called a “superficial pyoderma” or can be located in the lower layers of the skin (dermis), causing nodular abscesses in the skin (furunculosis) and draining tracts, in which case it is called a “deep pyoderma”
- Likely to recur or not be responsive to medical treatment, if underlying cause is not identified and managed effectively

ABNORMAL THICKENING IN THE LINING OF THE UTERUS, WITH THE PRESENCE OF FLUID-FILLED SACS (CYSTIC ENDOMETRIAL HYPERPLASIA) AND PUS IN THE UTERUS (PYOMETRA)



BASICS

OVERVIEW

- Cystic endometrial hyperplasia—hormonally mediated, progressive, abnormal thickening in the lining of the uterus (known as the “endometrium”), characterized by the presence of fluid-filled sacs or cysts
- Pyometra—develops secondary to cystic endometrial hyperplasia; develops when bacteria invade the abnormally thickened lining of the uterus (endometrium) and pus accumulates in the uterus
- The female dog is a “bitch;” the female cat is a “queen”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Usually greater than 6 years of age
- Young animals—especially if treated with female hormones (estrogen) or progestogen (any substance capable of producing the effects of the female hormone, progesterone)
- Dogs—usually diagnosed 1 to 12 weeks after “heat” or “estrus”
- Cats—onset relative to “heat” or “estrus” more variable
- Accumulation of pus in the uterine stump (known as “pyometra of the uterine stump”) in spayed animals—may develop any time after surgical removal of the ovaries and uterus (known as a “spay” or “ovariohysterectomy”)

Predominant Sex

- Female only

SIGNS/OBSERVED CHANGES in the ANIMAL

- Closed cervix (the “cervix” is the lower part of the uterus that extends into the vagina [the tubular passageway or birth canal, leading from the opening of the vulva to the cervix]; a “closed cervix” is one in which the muscles surrounding the cervix are contracted and the opening into the uterus is “shut” so no pus or discharge can drain from the uterus—signs of generalized (systemic) illness, progressing to signs of generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”) and shock
- Uterus—enlarged with closed cervix (where the pus or discharge cannot drain from the uterus); may not be enlarged with open cervix (where the muscles surrounding the cervix are relaxed, allowing the opening into the uterus to expand and pus or discharge to drain from the uterus, through the vagina and vulva [external genitalia])
- Discharge from the vulva (external genitalia)—depends if cervix is open or closed; discharge may be bloody or may contain mucus and pus
- Depression and sluggishness (lethargy)
- Lack of appetite (known as “anorexia”)
- Increased urination (known as “polyuria”) and increased thirst (known as “polydipsia”)
- Vomiting
- Abdominal distension

CAUSES

- Dogs—repeated exposure of the lining of the uterus (endometrium) to estrogen followed by exposure to progesterone with each “heat” or “estrus” (whether or not the bitch becomes pregnant); this hormonal pattern is unique to the bitch
- Cats—may be the result of estrogen at “heat” or “estrus,” followed by a progestational phase, caused by induction of release of eggs from the ovaries (ovulation) through breeding, spontaneous ovulation, or other (as yet undefined) stimuli

RISK FACTORS

- Old, intact females that have never given birth may be susceptible

- Pharmacologic use of estrogen (“mismatch”) shots during particular times of the “heat” or “estrous” cycle
- No correlation with “false pregnancy” or “pseudopregnancy” in dogs (in other words, a bitch with a history of false pregnancy is not at greater risk of developing cystic endometrial hyperplasia and/or pyometra than a bitch that has not had a false pregnancy)



TREATMENT

HEALTH CARE

- Inpatient
- Accumulation of pus in the uterus (pyometra)—life-threatening condition if the cervix is closed (where pus or discharge cannot drain from the uterus)
- Supportive care—immediate intravenous fluid administration and antibiotics

SURGERY

- Accumulation of pus in the uterus (pyometra), open and closed cervix—surgical removal of the ovaries and uterus (spay or ovariectomy) preferred treatment; long-term (chronic) progressive disease
- Accumulation of pus in the uterus (pyometra), with a closed cervix (where pus or discharge cannot drain from the uterus)—caution should be used during surgical removal of the ovaries and uterus (spay or ovariectomy); the enlarged uterus may be very fragile
- Uterine rupture or leakage of pus from the uterine stump—repeated flushing (lavage) of the abdominal cavity with sterile saline



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antibiotics

- Empirical, pending results of bacterial culture and sensitivity test
- Should be administered to all patients with inflammation characterized by accumulation of pus in the uterus (pyometra)
- Common choices—ampicillin; enrofloxacin (Baytril®)
- Antibiotics—not effective as the sole treatment, unless the uterus is of normal size and the serum progesterone level is less than 2 ng/ml on blood tests

Prostaglandins

Prostaglandin F_{2α} (PGF_{2α})

- Prostaglandins cause smooth muscle contractions and subsequent emptying of the uterus and decrease in serum progesterone concentration, based on dosage
- Dogs—for 2 to 7 days, until the uterus nears normal size as determined by feeling the abdomen, X-rays, or ultrasound examination or until no fluid is visible within the uterus as seen by ultrasound examination
- Cats—for 2 to 5 days, until the uterus nears normal size
- Re-evaluate patient 2 to 4 weeks after discontinuation of prostaglandins; if the uterus has increased in size or the patient still has marked discharge from the vulva (external genitalia), medical treatment protocol can be repeated
- Surgical removal of the ovaries and uterus (spay or ovariectomy)—performed in patients that do not respond to treatment with prostaglandin (lack of response indicated by continued enlargement of the uterus or presence of fluid in the uterus, or discharge from the vulva is still present after two courses of medical treatment)

Cloprostenol

- Dogs—injection under the skin (subcutaneous or SC injection) daily for 7 to 14 days

Miscellaneous

- Aglepristone—effectiveness improved with prostaglandin treatment at the same time; not readily available in the United States
- Cabergoline—for 7 to 14 days with prostaglandin treatment
- Drugs that enhance the immune response (such as estrogens) or certain agents (such as oxytocin and ergot), which induce contraction of the muscles of the uterus are unreliable; “oxytocin” is a female hormone that causes uterine contractions and promotes milk release during lactation



FOLLOW-UP CARE

PATIENT MONITORING

- Release from the hospital when the uterus is of near normal size or when no fluid is visible within the uterus by ultrasound examination, and clinical signs have lessened in severity or disappeared; re-evaluate in 2 to 4 weeks
- Antibiotics—administration should be continued for 3 to 4 weeks
- Discharge from the vulva (external genitalia)—may persist for up to 4 weeks
- Serial complete blood counts (CBCs)—the white-blood cell count rises rapidly after surgical removal of the ovaries and uterus (spay or ovariectomy), because the bone marrow continues to release neutrophils (a type of white-blood cell) into the bloodstream, from which they can no longer enter the uterus

PREVENTIONS AND AVOIDANCE

- Next proestrus (time from beginning of vaginal bleeding of “heat” until the bitch allows male to mount and breed)—obtain a specimen of the anterior vagina for bacterial culture, using a guarded culture swab
- Treat bitch with an appropriate antibiotic for 3 weeks
- Breed during the “heat” or “estrus” immediately following medical treatment—the pregnant uterus may be less susceptible to re-infection; bitch with underlying cystic endometrial hyperplasia has limited breeding life (best to get desired number of puppies as soon as possible); bitch not more likely to clear the disease spontaneously if allowed to cycle without being bred

POSSIBLE COMPLICATIONS

- Bitch may enter “heat” or “estrus” sooner after medical treatment than anticipated

EXPECTED COURSE AND PROGNOSIS

- Dogs—underlying cystic endometrial hyperplasia (thickening of the uterine lining with the presence of fluid-filled sacs or cysts) still exists; predisposed to recurrence
- Breed patient to desired stud dogs in a timely manner; use of subfertile stud dogs is not recommended
- Recommend surgical removal of ovaries and uterus (spay or ovariectomy) as soon as breeding life is over
- Closed-cervix (where pus or discharge cannot drain from the uterus) inflammation of the uterus with accumulation of pus (pyometra) can be associated with uterine rupture and inflammation of the lining of the abdomen (known as “peritonitis”)



KEY POINTS

- Surgical removal of ovaries and uterus (spay or ovariectomy) is the preferred treatment
- Medical treatment should be considered only for a valuable breeding animal that has an open cervix (where pus or discharge can drain from the uterus) and does not have excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”); except when breeding is planned, non-progestational, estrus-suppressing drugs must be given for life
- Medical treatment of closed-cervix (where pus or discharge cannot drain from the uterus) inflammation of the uterus with accumulation of pus (pyometra) can be associated with uterine rupture and inflammation of the lining of the abdomen (peritonitis)
- Medical treatment probably does not cure underlying cystic endometrial hyperplasia (thickening of the uterine lining with the presence of fluid-filled sacs or cysts) in patients with either open- or closed-cervix pyometra, but may enable some affected bitches to reproduce

PYOTHORAX

(PUS IN THE PLEURAL SPACE, THE SPACE BETWEEN THE CHEST WALL AND THE LUNGS)



BASICS

OVERVIEW

- Accumulation of pus within the pleural space (the space between the chest wall and lungs, which is lined by the pleura), usually associated with infection

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Dogs—hunting and sporting breeds
- Cats—domestic shorthair

Mean Age and Range

- Dogs and cats—median age approximately 4 years

SIGNS/OBSERVED CHANGES in the ANIMAL

- History of fights or puncture wounds
- Often subtle signs in onset, with few clinical signs until late in the course of disease
- Breathing problems—often not severe, unless the disease is advanced
- Vomiting/diarrhea may be initial sign for which animal is presented to the veterinarian in 25% of canine cases
- Diminished activity
- Collapse after exercising and slow recovery
- Weight loss and partial lack of appetite (anorexia) may be the only clinical signs
- Temporary improvement with antibiotic therapy
- Rapid breathing (known as “tachypnea”)—usually apparent; may be mild and not associated with difficulty breathing (known as “dyspnea”)
- Extreme weight loss with muscle wasting (known as “cachexia”)—often observed
- Cough—may be observed
- Fever (known as “pyrexia”)—usually low-grade, may be observed
- Muffled heart sounds, decreased lung sounds in the lower part of the chest, and increased lung sounds in the upper part of the chest (along the back) may be detected when listening to the heart and lungs with a stethoscope (known as “thoracic auscultation”)
- Cats—may show few clinical signs before onset of apparently sudden (acute) severe breathing difficulties (known as “respiratory distress”), collapse, and shock associated with generalized bacterial infection (sepsis; condition known as “septic shock”)
- Injury to the chest wall—may not be apparent or may be healed at the time of examination

CAUSES

- Infectious—dogs: bacteria including *Actinomyces*, *Nocardia*, anaerobes (bacteria that can live and grow in the absence of oxygen, such as *Bacteroides*, *Peptostreptococcus*, *Fusobacterium*), *Corynebacterium*, *Escherichia coli*, *Pasteurella*, and *Streptococcus*; fungal agents
- Infectious—cats: bacteria normally found in the mouth (such as *Pasteurella multocida* and *Bacteroides*) most common; obligate anaerobes (bacteria that must live and grow in the absence of oxygen, such as *Peptostreptococcus*, *Fusobacterium*) common
- Parasitic—dogs: esophageal rupture of *Spirocerca lupi* granuloma; *Spirocerca lupi* is a parasitic worm that lives in nodules in the esophagus; the nodules are known as “granulomas”
- Tumors or cancer—rarely with tumors in the chest, secondary to death of tumor tissue (known as “tumor necrosis”)
- Twisting of a lung lobe (known as “lung-lobe torsion”)—occasionally associated with accumulation of pus in the pleural space (pyothorax)
- Foreign bodies are found rarely, even with surgical exploration

RISK FACTORS

- Dogs—hunting, field trials, and other strenuous outdoor sporting activities
- Dogs—*Spirocerca lupi* should be considered in areas where the parasite is common (known as “endemic areas”)
- Cats—multi-cat household, outdoor cats, pneumonia, upper-respiratory infection



TREATMENT

HEALTH CARE

- Inpatient—often for several days to weeks
- Treat like any abscess; drainage is critical, without which resolution is highly unlikely
- Continuous removal or evacuation of pus from the pleural space (the space between the chest wall and lungs) via a tube inserted into an opening into the chest (known as “tube thoracostomy”) with low-pressure suction
- Cats—usually require general anesthesia for chest-tube placement
- Dogs with severe breathing problems—may use local anesthesia and regional nerve blocks to prevent pain (analgesia), rather than general anesthesia, for chest-tube placement
- Flushing the pleural space (the space between the chest wall and lungs) with fluid (known as “thoracic lavage”)—every 6 to 8 hours with warm, sterile saline; may help break down consolidated debris and accumulated pus
- Rapid, repeated blows or taps to the chest (known as “coupage” or “rapid thoracic percussion”)—may help remove consolidated debris

ACTIVITY

- Inpatient—encourage the patient to exercise lightly (10 minutes every 6 to 8 hours); promotes breathing efforts and helps break down developing scar tissue in the pleural space (the space between the chest wall and the lungs; scar tissue is known as a “pleural adhesion”)
- After discharge, gradually increase exercise over 2 to 4 months

DIET

- High-calorie food
- Protein replacement usually is unnecessary

SURGERY

- Surgical exploration, surgical removal of dead tissue and any foreign material (known as “débridement”) and potential surgical removal of a lung lobe (known as “lobectomy”) may be required in some cases and can ensure a better outcome than with medical treatment alone
- Surgery—likely to be associated with higher cure rate if lung abscesses are present, if scar tissue has developed in the pleural space (pleural fibrosis), if a lung lobe has twisted (lung-lobe torsion), if extensive development of pockets of pus is present, or if the mediastinum (the center portion of the chest that contains the heart and other organs [except for the lungs]) is involved
- Identified foreign body via chest imaging (such as X-rays, ultrasound, computed tomography or CAT scan [CT], or magnetic resonance imaging [MRI])—surgical incision into the chest (known as “thoracotomy”) and retrieval of the foreign body is indicated; grass awns are found rarely, even during surgery; attempted surgical retrieval is not recommended unless foreign body is visualized via imaging



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antibiotics

- Ultimately, choice determined by bacterial culture and sensitivity testing
- Suspected specific pathogen—initiate treatment before bacterial culture and sensitivity results are available; choose on the basis of common antibiotic sensitivities of particular organisms; *Actinomyces* and *Bacteroides* (but not *Bacteroides fragilis*) often are susceptible to amoxicillin; *Nocardia* often are susceptible to potentiated sulfonamides; obligate anaerobic bacteria (bacteria that must live and grow in the absence of oxygen including, *Bacteroides fragilis*) are susceptible to amoxicillin–clavulanic acid, chloramphenicol, and usually metronidazole; *Pasteurella* often are susceptible to potentiated penicillins
- Ampicillin or amoxicillin with a β -lactamase inhibitor—good initial choice for most patients; ampicillin and sulbactam followed by amoxicillin–clavulanic acid, when medications can be given by mouth
- Trimethoprim-sulfa, aminoglycosides, and quinolones—generally ineffective

- Multiple antibiotics occasionally necessary
- Dosages are generally high, to allow adequate distribution into the pleural space (the space between the chest wall and the lungs); may need to continue drug for several months and occasionally indefinitely

Pain Relievers (Analgesics)

- May be required following surgical incision into the chest (thoracotomy) or during procedure to tap the chest and to remove fluid from the pleural space (the space between the chest wall and lungs; procedure known as “pleurocentesis”)
- With severe discomfort—may use fentanyl patch or intravenous/intramuscular agents
- Effectiveness of drugs to decrease pain and/or anesthetize the surfaces of the lining of the chest (known as “intrapleural anesthesia,” such as bupivacaine [a local anesthetic] mixed with the fluid to flush the pleural space [lavage fluid]) may be limited by the presence of pus in the pleural space (the space between the chest wall and lungs)



FOLLOW-UP CARE

PATIENT MONITORING

- Measure net chest-fluid production—determine when chest tubes may be removed
- Periodic chest X-rays—to ensure proper chest-tube placement, and lack of pocketing of pus in localized areas; to determine whether an additional chest tube should be placed on the opposite side of the chest; to determine if primary lung disease exists, that may not have been apparent on initial examination
- Evaluate chest X-rays—ensure adequate removal of fluid from the pleural space (the space between the chest wall and lungs)
- Assess complete blood count (CBC) and chest X-rays monthly—residual changes seen on X-rays may be permanent, but fluid should be absent
- Repeat bacterial culture and sensitivity testing, if the patient fails to improve
- Antibiotics—continue for 1 month after the patient is clinically normal, the complete blood count (CBC) is normal, and no evidence of fluid re-accumulation is seen on X-rays; average duration of therapy is 3 to 4 months, but may continue for 6 to 12 months or longer

PREVENTIONS AND AVOIDANCE

- Avoid activity that increases the likelihood that the animal will develop pyothorax (“pus in the chest”)—often not practical

POSSIBLE COMPLICATIONS

- Problems with the chest tube—may prevent adequate drainage or produce pneumothorax (accumulation of free air in the pleural space, the space between the chest wall and lungs); the chest tube may put pressure on the arteries and veins to the front leg, resulting in fluid build-up (known as “limb edema”) or lameness; injury to the lung itself during placement of the chest tube
- Persistent, recurrent accumulation of pus in the pleural space (pyothorax)—compartmentalization of pus; premature discontinuation of treatment; lung lesions
- Long-term (chronic) inflammation of the pleura (the lining of the pleural space), characterized by scar tissue (known as “fibrosing pleuritis”) and poor performance after apparent recovery—may occasionally respond to surgery
- Persistent inflammation of the mediastinum (the center portion of the chest that contains the heart and other organs [except for the lungs]; inflammation is known as “mediastinitis”)

EXPECTED COURSE AND PROGNOSIS

- With aggressive management—prognosis fair to excellent (60% to 90% survival)
- With repeated intermittent antibiotic therapy only or with inadequate drainage—prognosis poor
- Return to performance—depends on long-term duration of disease and level of management



KEY POINTS

- Duration of treatment (inpatient and outpatient) is long and expensive; average duration of therapy is 3 to 4 months, but may continue for 6 to 12 months or longer
- With aggressive management—prognosis fair to excellent (60% to 90% survival)

PYTHIOSIS

(INFECTION WITH THE WATER MOLD, *PYTHIUM*)



BASICS

OVERVIEW

- An infectious disease affecting primarily the gastrointestinal tract or skin of dogs and cats
- It is caused by the water mold, *Pythium insidiosum*

GENETICS

- Although large-breed dogs are affected most often, no genetic basis has been documented

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Cats—less commonly

Breed Predilection

- Large-breed dogs, especially those used in hunting or field trial work near water
- Labrador retrievers
- German shepherd dogs may be more likely than other breeds to develop infection in the skin (known as “cutaneous pythiosis”)

Mean Age and Range

- Animals less than 3 years of age are most likely to be infected

Predominant Sex

- Males are affected more often than females, possibly because of increased exposure

SIGNS/OBSERVED CHANGES in the ANIMAL

- Affected dogs usually are not severely ill, until late in the course of disease
- Long-term (chronic) weight loss and intermittent vomiting are the most common signs
- Diarrhea may be evident, if the large intestine (colon) or a large segment of the small intestine is affected
- Regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth) is noted with rare esophageal disease
- Skin (cutaneous) disease characterized by nodules that ulcerate and drain

Gastrointestinal Pythiosis

- Affected dogs usually are bright and alert
- Severe weight loss; emaciation is common
- An abdominal mass often is felt by the veterinarian during physical examination
- Fever is noted occasionally
- Generalized (systemic) signs and abdominal pain may occur with intestinal blockage or obstruction, sudden lack of blood supply that leads to death of intestinal tissues (known as “infarction”), or abnormal opening or hole in the intestines (known as a “perforation”)

Skin (Cutaneous) Pythiosis

- Skin (cutaneous) lesions or lesions under the skin (known as “subcutaneous lesions”) appear as nonhealing wounds; boggy, fluid-filled regions; or poorly defined nodules that become ulcerated
- Multiple draining tracts with blood-tinged discharge or pus often are present

CAUSES

- *Pythium insidiosum*

RISK FACTORS

- Environmental exposure to swampy areas, bayous, ponds, or lakes containing infective *Pythium* spores
- Outdoor activities, such as hunting
- Dependent on geographic distribution—in the United States, pythiosis occurs most often in states bordering the Gulf of Mexico; however, it has been documented in Oklahoma, Arkansas, Missouri, Kentucky, Tennessee, North and South Carolina, Virginia, southern

Indiana, and New Jersey; outside the United States, pythiosis has been reported in Australia, Brazil, Burma, Colombia, Costa Rica, Indonesia, Japan, New Guinea, and Thailand



TREATMENT

HEALTH CARE

- The treatment of choice is aggressive surgical removal of all infected tissue; unfortunately, many animals are not presented to the veterinarian until late in the disease, when complete removal is not possible
- Supportive care may include fluids, potassium, nutritional support, and antibiotics (as needed)

ACTIVITY

- Limit activity

DIET

- Feed a highly digestible, calorie-dense diet

SURGERY

- Attempt wide surgical removal of infected tissue, even if medical treatment is contemplated
- Amputation is recommended for treatment of lesions involving the legs
- Enlarged abdominal lymph nodes should be biopsied
- Dogs often improve after lesions causing blockage or obstruction of the gastrointestinal tract are removed surgically, even if significant disease still is present
- Postoperative medical treatment with itraconazole and terbinafine for 2 to 3 months is recommended to decrease the chance of recurrence



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Itraconazole combined with terbinafine (both antifungal medications) appears to be the most effective medical treatment
- Medical treatment should be continued for a minimum of 6 months
- Give itraconazole with food
- Amphotericin-B lipid complex has shown effectiveness in a limited number of dogs with gastrointestinal pythiosis; its use is recommended when the patient cannot tolerate medications administered by mouth



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor for signs of recurrence
- ELISA serologic tests (blood tests that detect the presence of antigens [substances that induce an immune response], of a certain disease-causing agent, in this case *Pythium*) can be used to monitor response to therapy; serology should be checked 2 to 3 months after surgery, or every 3 months during medical treatment
- Abdominal ultrasound is useful in re-evaluating intestinal lesions
- Medical management may reduce the extent of large lesions to the point that they become removable surgically
- Blood tests (liver enzymes) should be evaluated monthly while patient is on itraconazole
- Blood tests (serum blood urea nitrogen, creatinine, and potassium) should be evaluated before each dose of amphotericin-B lipid complex is administered

POSSIBLE COMPLICATIONS

- Sudden (acute) severely painful abdomen and death from gastrointestinal blood clots (medical term for blood clot is “thrombosis”) and abnormal opening or hole in the stomach or intestines (known as a “perforation”)

EXPECTED COURSE AND PROGNOSIS

- Prognosis is guarded to poor, unless complete surgical removal of infected tissue is possible
- Less than 10% of affected animals are cured with medical treatment alone
- Re-evaluation of ELISA serologic tests (blood tests that detect the presence of antigens [substances that induce an immune response] of a certain disease-causing agent, in this case *Pythium*) 2 to 3 months after surgery is an excellent prognostic indicator



KEY POINTS

- Treatment is expensive
- Prognosis is guarded to poor, unless a complete surgical removal of infected tissue is possible

RABIES



BASICS

OVERVIEW

● A severe, invariably fatal, viral inflammation of the gray matter of the brain (known as “polioencephalitis”) of warm-blooded animals, including humans; “gray matter” is the nerve tissue of the brain that contains the nerve cell bodies

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- All warm-blooded animals, including dogs, cats, and humans
- United States—four strains of rabies virus are found in the skunk, raccoon, bat, and fox populations; all four strains can be transmitted to dogs and cats

Mean Age and Range

- None, but adult animals that come in contact with wildlife are at most risk

SIGNS/OBSERVED CHANGES in the ANIMAL

- Quite variable; atypical presentation is the rule rather than the exception
- Three progressive stages of disease—1) prodromal stage—early signs of disease; signs may include change in behavior, apprehension, nervousness, seeking solitude; 2) furious stage—signs may include irritability, excitability, avoidance of light (known as “photophobia”), and viciousness (biting, attacking); and 3) paralytic stage—also known as the “dumb form” of rabies; signs may include paralysis of various parts of the body (determined by location of original site of exposure to the rabies virus, such as a bite wound), change in voice (known as “dysphonia”), excessive salivation/drooling, and choking sounds; final signs include coma and death
- Change in attitude—animal seeks solitude; apprehension, nervousness, anxiety; unusual shyness or aggressiveness
- Erratic behavior—biting or snapping; licking or chewing at site of wound; biting at cage; wandering and roaming; excitability; irritability; viciousness
- Disorientation
- Muscular incoordination; seizures; inability to move voluntarily (known as “paralysis”)
- Change in tone of bark
- Excess salivation or frothing
- Paralysis of the lower jaw (mandible) and voice box or larynx; dropped jaw
- Inability to swallow
- Fever

CAUSES

- Rabies virus

RISK FACTORS

- Exposure to wildlife, especially skunks, raccoons, bats, and foxes
- Lack of adequate vaccination against rabies
- Bite or scratch wounds from unvaccinated dogs, cats, or wildlife
- Exposure to aerosols in bat caves
- Animals that do not have the ability to develop a normal immune response (known as an “immunocompromised animal”)—use of modified live virus rabies vaccine



TREATMENT

HEALTH CARE

- Strictly inpatient for animal suspected of being exposed to rabies or having rabies

- Administer nursing care with extreme caution
- No treatment for rabies
- Once the diagnosis is certain, euthanasia is indicated

ACTIVITY

- Confine to secured quarantine area with clearly posted signs indicating suspected rabies
- Runs or cages should be locked; only designated people should have access
- Feed and water without opening the cage or run door (in other words, pass food and water bowls into the cage or run through specialized access points designed for such use)

DIET

- Soft, moist food; most patients will not eat

SURGERY

- Generally none
- Skin biopsy—may help establish diagnosis before death of the animal; diagnosis must be confirmed by identification of rabies virus infection from central nervous system tissue



FOLLOW-UP CARE

PATIENT MONITORING

- All suspected rabies patients should be isolated securely and monitored for any development of mood change, attitude change, or clinical signs that might suggest the diagnosis
- An apparently healthy dog or cat that bites or scratches a person should be monitored for a period of 10 days or according to local or state regulations; if no signs of illness occur in the animal within 10 days, the person has had no exposure to the virus; dogs and cats do not shed the virus for more than 3 days before development of clinical disease
- An unvaccinated dog or cat that is bitten or exposed to a known rabid animal must be quarantined for up to 6 months or according to local or state regulations

PREVENTIONS AND AVOIDANCE

- Vaccines (dogs and cats)—vaccinate according to standard recommendations and state and local requirements; all dogs and cats with any potential exposure to wildlife or other dogs and cats; vaccinate after 12 weeks of age; then 12 months later; then every 3 years using a vaccine approved for 3 years' duration; use only inactivated virus or recombinant vector vaccines for cats
- Rabies-free countries—entering dogs and cats are quarantined for long periods, usually 6 months
- Disinfection—any contaminated area, cage/run, food dish, water bowl or instruments must be disinfected thoroughly; use a 1:32 dilution (4 ounces per gallon) of household bleach to inactivate the virus quickly

POSSIBLE COMPLICATIONS

- Paralysis
- Attitude or behavior changes
- Death
- Exposure of rabies virus to other animals or people

EXPECTED COURSE AND PROGNOSIS

- Prognosis—grave; almost invariably fatal
- All dogs and cats with clinical infection will succumb within 7 to 10 days of onset of clinical signs



KEY POINTS

- Rabies is a serious, usually fatal infection for the animal; rabies can be spread from animals to people (known as having “zoonotic potential”)
- Tell your veterinarian about any possible human exposure (such as contact with the animal or a bite or scratch)

- Any person possibly exposed to rabies should see a physician immediately
- Local public health officials must be notified

RED EYE



BASICS

OVERVIEW

- Redness (known as “hyperemia”) of the eyelids or involving blood vessels in the eye or moist tissues (conjunctiva) of the eye, or bleeding within the eye

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on cause
- May affect one or both eyes
- Result of generalized (systemic) disease—signs associated with abnormalities in other organ systems

CAUSES

- Virtually every case fits into one or more of the following categories:
 - Inflammation of the eyelids (known as “blepharitis”)
 - Inflammation of the moist tissues of the eye (known as “conjunctivitis”)
 - Inflammation of the cornea (the clear outer layer of the front of the eye; condition known as “keratitis”)
 - Inflammation of the white, firm, outer portion of the eyeball (known as “scleritis”)
 - Inflammation of the front part of the eye, including the iris (known as “anterior uveitis”)
 - Disease of the eye, in which the pressure within the eye is increased (known as “glaucoma”)
 - Blood in the anterior chamber of the eye (the front part of the eye, between the cornea and the iris; accumulation of blood known as “hyphema”)
 - Disease of the bony cavity containing the eyeball (known as “orbital disease”)—usually the orbital abnormality is more prominent than the “red eye”

RISK FACTORS

- Generalized (systemic) infectious or inflammatory diseases
- Lack of capacity to develop an immune response (known as “immunocompromise”)
- Blood-clotting disorders (known as “coagulopathies”)
- Generalized (systemic) high blood pressure (known as “hypertension”)
- Irritation by medications applied to the eye directly (known as “topical ophthalmic medications”)—aminoglycosides; pilocarpine; epinephrine
- Cancer
- Trauma



TREATMENT

HEALTH CARE

- Usually outpatient
- Elizabethan collar may be used to prevent self-trauma
- Avoid dirty environments or those that may lead to eye trauma, especially if topical (applied to the eye directly) steroids are used
- Consider referral to a veterinary ophthalmologist (eye specialist)
- Few causes of “red eye” are fatal; however, a workup may be indicated (especially with inflammation of the front part of the eye, including the iris [anterior uveitis] and blood in the anterior chamber of the eye [hyphema]) to rule out potentially fatal generalized

(systemic) diseases

SURGERY

- Deep corneal ulcers (loss of tissue on the surface of the cornea [the clear part of the front of the eye]); and glaucoma (condition where pressure within the eye is increased)—surgical treatment may be best



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Depend on specific cause
- Generally, control eye pain, inflammation, infection, and intraocular pressure (IOP); increased IOP indicates glaucoma
- Aspirin—may control mild eye pain and inflammation; use as directed by your pet’s veterinarian
- Flunixin meglumine—may be used in dogs with severe eye inflammation



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on cause
- Repeat eye examinations as required to ensure that eye pain, inflammation, and intraocular pressure (IOP) are well controlled
- The greater the risk of loss of vision, the more closely the patient needs to be followed; may require daily or more frequent examination

PREVENTIONS AND AVOIDANCE

- Depend on cause

POSSIBLE COMPLICATIONS

- Loss of the eye or permanent vision loss
- Long-term (chronic) eye pain and inflammation
- Death, if “red eye” is secondary to a generalized (systemic) disease process

EXPECTED COURSE AND PROGNOSIS

- Depend on cause



KEY POINTS

- Redness (known as “hyperemia”) of the eyelids or involving blood vessels in the eye or moist tissues (conjunctiva) of the eye, or bleeding within the eye
- May affect one or both eyes
- Possible complications include loss of the eye or permanent vision loss; long-term (chronic) eye pain and inflammation; and in some cases, death (if “red eye” is secondary to a generalized [systemic] disease process)

REGURGITATION

(RETURN OF FOOD OR OTHER CONTENTS FROM THE ESOPHAGUS, BACK UP THROUGH THE MOUTH)



BASICS

OVERVIEW

- Passive, backward movement or return of food or other contents from the esophagus (part of the digestive tract, the tube running from the throat to the stomach) into the throat (pharynx) or mouth

GENETICS

- Regurgitation due to an enlarged esophagus (known as “megaesophagus”) can be inherited in wire fox terriers (autosomal recessive) and miniature schnauzers (autosomal dominant or 60% penetrance autosomal recessive)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs (more commonly) and cats

Breed Predispositions

- Dogs—wire fox terriers, miniature schnauzers; other susceptible breeds include Great Danes, German shepherd dogs, Irish setters, Labrador retrievers, Newfoundlands, Chinese shar peis
- Cats—Siamese and Siamese-related

Mean Age and Range

- Congenital (present at birth) cases are identified soon after birth, or at weaning from liquid to solid foods
- Acquired (present later in life/after birth) cases may be seen at any age, depending on the cause

Predominant Sex

- No predominant sex has been identified as being more likely to regurgitate

SIGNS/OBSERVED CHANGES in the ANIMAL

- Owners often report “vomiting;” veterinarian must differentiate vomiting (forceful ejection of stomach contents up through the esophagus and mouth) from regurgitation (passive, backward movement or return of food or other contents from the esophagus [part of the digestive tract, the tube running from the throat to the stomach] into the throat [pharynx] or mouth)
- Regurgitation—passive; little to no abdominal effort; regurgitated material has increased amounts of thick mucus in it
- Vomiting—active process; forceful ejection of stomach contents up through the esophagus and mouth; vomited material has increased bile staining (yellowish staining) in it
- The shape of the expelled material, presence of undigested food, and length of time from ingestion to regurgitation or vomiting are less helpful to differentiate between regurgitation and vomiting
- Difficulty swallowing (known as “dysphagia”)
- Coughing
- Ravenous appetite
- Weight loss; may be extreme weight loss with muscle wasting (known as “cachexia”)
- Other signs, depending upon underlying cause
- Swelling may be noted in the neck
- Excessive salivation/drooling (known as “ptyalism”)
- Bad breath (known as “halitosis”)
- Increased breathing noises
- Discharge from the nose
- Fever, if animal also has pneumonia
- Weakness

CAUSES

Congenital Pharyngeal Problems (problems involving the throat, present at birth)

- Cleft or short palate

- Cricopharyngeal achalasia (neuromuscular disorder of young dogs in which the function of the muscles in the throat and upper esophagus is abnormal, leading to swallowing difficulties)
- Myasthenia gravis (a disorder of neuromuscular transmission characterized by muscular weakness and excessive fatigue)

Congenital Esophageal Problems (problems involving the esophagus, present at birth)

- Persistent right aortic arch (abnormal development of major arteries of the heart, resulting in the esophagus being trapped by the blood vessels causing obstruction)
- Enlarged esophagus (megaesophagus)
- Glycogen-storage diseases (inherited diseases in which normal glycogen [the body's carbohydrate reserve] metabolism is altered)
- Diverticulum (a pouch or sac, opening from the esophagus)
- Abnormal opening between a bronchus (airway) and the esophagus (known as "bronchoesophageal fistula")

Acquired Pharyngeal Problems (problems involving the throat, develop later in life)

- Foreign bodies
- Cancer
- Rabies
- Poisoning or toxicity (botulism)
- Muscle disease (known as "myopathy") and/or nervous system disease (known as "neuropathy")

Acquired Esophageal Problems (problems involving the esophagus, develop later in life)

- Enlarged esophagus (megaesophagus)
- Myasthenia gravis (a disorder of neuromuscular transmission characterized by muscular weakness and excessive fatigue)
- Abnormal narrowing of the esophagus (known as a "stricture")
- Tumor or cancer
- Hormonal or endocrine disease
- Hiatal hernia
- Folding of part of the stomach into the esophagus (the tube running from the throat to the stomach; condition known as "gastroesophageal intussusception")
- Backward or reverse flow of stomach contents into the esophagus (known as "gastroesophageal reflux")
- Tumors around the esophagus
- Dysfunction of the autonomic nervous system (known as "dysautonomia")
- Muscle disease (myopathy) and/or nervous system disease (neuropathy)
- Foreign bodies
- Disease characterized by the formation of nodules (known as "granulomatous disease") involving the esophagus
- Poisoning or toxicity (lead)
- Unknown cause (known as "idiopathic" disease)
- Stomach dilates with gas and/or fluid (known as "gastric dilatation"), and subsequently rotates around its short axis (known as "volvulus")—condition known as "gastric dilatation-volvulus" or "bloat")
- Parasitic infection—*Spirocerca lupi*; *Spirocerca lupi* is a parasitic worm that lives in nodules in the esophagus; the nodules are known as "granulomas"
- Abnormal opening between a bronchus (airway) and the esophagus (known as "bronchoesophageal fistula")

RISK FACTORS

- Possible risk of backward or reverse flow of stomach contents into the esophagus (gastroesophageal reflux) with general anesthesia



TREATMENT

HEALTH CARE

- Therapy for underlying cause should be instituted
- Most important are meeting nutritional requirements and preventing or treating aspiration pneumonia
- Aspiration pneumonia may require oxygen therapy; administration of medication in a fine spray (known as "nebulization") and efforts to dislodge secretions in the lungs and to induce coughing (known as "coupage"); fluid therapy with balanced electrolyte solution
- These animals may be recumbent and require soft bedding and should be maintained up on their chests (in sternal recumbency) or turned to alternate down side every 4 hours

ACTIVITY

- Depending on cause, restricted activity usually is not necessary

DIET

- Experiment with different food consistencies—liquid gruel, small meatballs, food slurries made by using a kitchen blender
- Some cases benefit from tube feeding
- Food and water should be elevated, and the animal should be maintained in an upright position 10 to 15 minutes after eating or drinking

SURGERY

- Surgical intervention is indicated for treatment of abnormal development of major arteries of the heart, resulting in the esophagus being trapped by the blood vessels causing obstruction (abnormal arteries known as a “vascular ring anomaly”); neuromuscular disorder of young dogs in which the function of the muscles in the throat and upper esophagus is abnormal, leading to swallowing difficulties (cricopharyngeal achalasia)—surgical treatment is a “cricopharyngeal myotomy;” abnormal opening between a bronchus (airway) and the esophagus (bronchoesophageal fistula); and other congenital (present at birth) lesions
- Balloon dilation (procedure in which an instrument with an expandable balloon is inserted into the esophagus and the balloon is expanded to open the narrowing or stricture) is indicated for cases of esophageal stricture



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics for aspiration pneumonia (broad spectrum or based on bacterial culture and sensitivity from transtracheal wash [TTW] or bronchoalveolar lavage [BAL]; TTW and BAL are techniques in which samples from within the respiratory tract are collected for evaluation by microscope and/or bacterial culture and sensitivity testing)
- Specific therapy for underlying cause, if indicated
- Drugs that improve the propulsion of contents through the stomach and intestines (known as “gastrointestinal prokinetic agents”)—metoclopramide may increase tone of the muscle between the stomach and esophagus (gastroesophageal sphincter), increases stomach motility, and may increase movement of the esophagus; cisapride is more effective for preventing backward or reverse flow of stomach contents into the esophagus (known as “gastroesophageal reflux”) than metoclopramide; however, it slows the time for materials to move through the esophagus (known as “esophageal transit time”); may be more helpful in cats due to increased smooth muscle in the lower esophagus
- Other motility agents (such as nizatidine) have not been evaluated for effects on esophageal motility
- H₂-blockers for inflammation of the esophagus (known as “esophagitis”)—ranitidine, cimetidine, famotidine



FOLLOW-UP CARE

PATIENT MONITORING

- Animals with aspiration pneumonia should have chest X-rays and complete blood counts (CBCs) checked until resolution, or if recurrence is suspected
- Animals should be monitored and weighed to ensure adequate caloric intake

POSSIBLE COMPLICATIONS

- Aspiration pneumonia
- Others depending on presence of other diseases (such as inadequate production of thyroid hormone [known as “hypothyroidism”])

EXPECTED COURSE AND PROGNOSIS

- An older animal with enlarged esophagus of unknown cause (known as “idiopathic megaesophagus”) has a poor prognosis
- Aspiration pneumonia is the typical cause of death or euthanasia



KEY POINTS

- Regurgitation is the passive, backward movement or return of food or other contents from the esophagus (part of the digestive tract, the tube running from the throat to the stomach) into the throat (pharynx) or mouth
- Most cases of enlarged esophagus (megaesophagus) require lifelong therapy, even if an underlying cause is found
- Client dedication is important for long-term management
- Most animals will succumb to aspiration pneumonia

RETINAL DEGENERATION



BASICS

OVERVIEW

- “Retinal” refers to the retina; the retina is the innermost lining layer (located on the back surface) of the eyeball; it contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision
- “Degeneration” is defined as a decline in function or structure
- “Retinal degeneration” is a decline in function or structure of the retina from any cause; the cause may be inherited or acquired (condition that develops sometime later in life/after birth)

GENETICS

- Hereditary—inherited retinal degeneration is more frequent in dogs than in cats
- Inherited—a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time (known as “progressive retinal atrophy” or “PRA”); may be subdivided into abnormal development of the light-sensitive cells of the retina (known as “photoreceptor dysplasias”), which begin before the retina fully develops (at less than 12 weeks of age), and decline in function or structure of the light-sensitive cells of the retina (known as “photoreceptor degenerations”), which begin after the retina is fully developed and mature

Dogs

- Progressive retinal atrophy (a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time)—autosomal recessive in most breeds, especially collies, Irish setters, miniature poodles, cocker spaniels, briards and Labrador retrievers; dominant in mastiffs, X-linked in Samoyeds and Siberian huskies
- Central progressive retinal atrophy (eye disease characterized by deterioration of the retina leading to loss of central vision, but retention of peripheral vision possibly for years)—autosomal dominant with incomplete penetrance in Labrador retrievers
- Inheritance in many breeds not determined
- Neuronal ceroid lipofuscinosis (a group of inherited, nervous system disorders with swelling and/or changes in the light-sensitive cells of the retina)—autosomal recessive (proven or presumed) in most breeds studied
- Inability to see clearly in bright light (known as “hemeralopia”)—autosomal recessive abnormal development of the light-sensitive cones in the retina (known as “cone dysplasia”) in Alaskan malamutes; undetermined inheritance in miniature poodles

Cats

- Abnormal development of the light-sensitive rods and cones in the retina (known as “rod-cone dysplasia”), Abyssinians have 2 forms—autosomal dominant: clinical signs at 4 months of age; autosomal recessive: may be blind by 2 years of age; also may have later onset of 2 years of age with vision problems by 4 years of age
- Isolated reports of both dominant and recessive inheritance in young Persians and domestic shorthairs
- Vision loss that becomes worse over time (known as “gyrate atrophy”)—autosomal recessive; caused by a build-up of a particular compound (ornithine) due to a lack of the enzyme that normally converts ornithine to glutamate (known as an “ornithine aminotransferase deficiency”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Abnormal development of the retina (retinal dysplasia) may be associated with Samoyeds and Labrador retrievers
- Retinal dysplasia also may be associated with multiple other eye abnormalities in Akitas and Doberman pinschers

Hereditary—Dogs (many breeds)

- Abnormal development of the retina (retinal dysplasia)—Bedlington terrier, Sealyham terrier, English springer spaniel, cocker spaniel
- Early-onset progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time)—Irish setter; collie; Norwegian elkhound; miniature schnauzer; Belgian shepherd, mastiff, Cardigan Welsh corgi, and briard (congenital [present at birth] stationary night blindness)
- Late-onset progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time)—miniature and toy poodle; American and English cocker spaniels; Labrador retriever; Tibetan terrier; miniature longhair dachshund; Akita; Samoyed; Siberian husky
- Central progressive retinal atrophy (eye disease characterized by deterioration of the retina leading to loss of central vision, but retention of peripheral vision possibly for years)—Labrador retriever; golden retriever; border collie; collie; Shetland sheepdog; briard
- Neuronal ceroid lipofuscinosis (a group of inherited, nervous system disorders with swelling and/or changes in the light-sensitive cells of

the retina)—English setter; Dalmatian; Tibetan terrier; collie

- Sudden blindness due to “sudden acquired retinal degeneration” or “SARD”—Brittany; miniature schnauzer; dachshund, any breed
Hereditary—Cats

- Abyssinian, Siamese, Persian, domestic shorthair

Mean Age and Range

- Early progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time)—3 to 4 months of age up to 2 years of age

- Late progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time)—clinical signs when the animal is greater than 4 to 6 years of age

- Sudden blindness due to sudden acquired retinal degeneration or SARD—middle-aged to old dogs

Predominant Sex

- Progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time)—none, except possibly X-linked recessive condition in Siberian huskies

- Sudden blindness due to sudden acquired retinal degeneration or SARD—70% are female

SIGNS/OBSERVED CHANGES in the ANIMAL

- Progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time) in dogs—gradually progressing night blindness (known as “nyctalopia”) that ultimately affects vision in bright light; may note dilated pupils or brighter tapetal reflex at night; may appear to be suddenly (acutely) blind (when patient finally becomes totally blind or is moved to unfamiliar surroundings)

- Inability to see clearly in bright light (hemeralopia)—rare; light-sensitive cones degenerate; day vision lost

- Central progressive retinal atrophy (eye disease characterized by deterioration of the retina leading to loss of central vision, but retention of peripheral vision possibly for years) in the dog—rare in the United States; central vision lost; may never become completely blind; may have difficulty locating stationary objects in bright light (especially hunting breeds)

- Sudden blindness due to sudden acquired retinal degeneration or SARD—vision lost in 1 to 4 weeks; increased urination (known as “polyuria”), increased thirst (known as “polydipsia”), and increased appetite (known as “polyphagia”) is common

- If severe retinal degeneration—light reflexes of the pupil are impaired or nearly abolished; the “pupil” is the circular or elliptical opening in the center of the iris of the eye; light passes through the pupil to reach the back part of the eye (known as the “retina”); the iris is the colored or pigmented part of the eye; the pupil constricts or enlarges (dilates) based on the amount of light entering the eye; the pupil constricts with bright light and enlarges in dim light—these actions are the “light reflexes of the pupil”

- Various changes in the appearance of the retina (light-sensitive lining of the back of the eye) may be noted when the veterinarian examines the back of the eye with an ophthalmoscope

- Progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time) in dogs—cataracts are common; a cataract is an opacity in the normally clear lens, if it is complete it prevents passage of light to the back part of the eye (retina)

- Sudden blindness due to sudden acquired retinal degeneration or SARD in dogs—obesity; enlargement of the liver (known as “hepatomegaly”); may note slow or absent light reflexes of the pupil; the pupil constricts or enlarges (dilates) based on the amount of light entering the eye; the pupil constricts with bright light and enlarges in dim light—these actions are the “light reflexes of the pupil”

CAUSES

Degenerative

- Progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time)—genetic; affects both eyes symmetrically

- Long-term (chronic) or uncontrolled glaucoma (disease of the eye, in which the pressure within the eye is increased)—decrease in tissue of the retina (known as “retinal atrophy;” the retina is the light-sensitive lining of the back of the eye) and optic nerve (the nerve that runs from the back of the eye to the brain)

- Secondary to scarring from separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid;” condition known as “retinal detachment”) or inflammation of the retina

Anomalous (abnormal structure)

- Abnormal development of the light-sensitive rods and cones of the retina (known as “rod-cone photoreceptor dysplasias”)—inherited disease; affects both eyes

- Other types of abnormal development (dysplasias)—may be located in multiple areas of the retina (so called “multifocal”) and non-blinding (for example, in English springer spaniels and Labrador retrievers)

Metabolic

- Mucopolysaccharidosis (disease related to a lack of or insufficient amount of a particular enzyme)—mixed-breed dogs; Siamese and domestic shorthairs (cats)

- Ornithine aminotransferase deficiency—a mitochondrial enzyme; progressive and total gyrate atrophy of the choroid and retina due to a build-up of a particular compound (ornithine) due to a lack of the enzyme that normally converts ornithine to glutamate

Cancer

- Cancer cells infiltrating the retina
- Scars from previous retinal detachment (separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball), if treated

Nutritional

- Severe deficiency of vitamin E or A (dogs and cats)—experimentally or dogs fed poor diets (high in polyunsaturated fats) may cause partial or complete degeneration of the retina
- Taurine deficiency (cats)—causes retinal degeneration and a heart-muscle disorder (known as “dilated cardiomyopathy”); taurine is an amino acid (protein) that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the retina

Infectious/Immune

- Infectious inflammation of the retina (known as “retinitis”) or inflammation of the choroid and retina (known as “chorioretinitis”); the “choroid” is located immediately under the retina and is part of the middle-layer of the eyeball that contains the blood vessels
- Infection may extend from or to the central nervous system (brain)**Idiopathic (Unknown Cause)**
- Sudden blindness due to sudden acquired retinal degeneration or SARD—dogs**Toxic**
- Individual pet is more likely to develop ill effects to a particular medication than other animals (known as “idiosyncratic reactions”)—griseofulvin or enrofloxacin (cats)
- Concurrent administration of ketamine hydrochloride and methylnitrosourea induces widespread (diffuse) retinal degeneration (cats)

RISK FACTORS

- Eye disease—cataracts; inflammation of the back part of the eye; inflammation of the choroid and retina (chorioretinitis); retinal detachment; glaucoma
- Taurine-deficient diet—dog food fed to cats (most cat foods now contain proper taurine levels)
- Genetics
- Cats—enrofloxacin (an antibiotic)



TREATMENT

DIET

- Cats—food should contain 500 to 750 ppm of taurine; taurine is an amino acid (protein) that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the retina
- Dogs—balanced diet; avoid all meat diet, high in polyunsaturated fats

SURGERY

- Not indicated in patients with blind, nonpainful eyes



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No medications currently are effective in treating retinal degeneration
- Pyridoxine supplementation (cats)—for ornithine aminotransferase deficiency; may increase activity of the enzyme; has not arrested or reversed degeneration of the retina
- Adequate dietary taurine—may halt the progression of the retinal deterioration due to inadequate levels of taurine (taurine-deficient retinopathy); taurine is an amino acid (protein) that is an important component of the diet of cats; cats cannot produce enough taurine in their bodies and so, must obtain taurine from their food to maintain the health of several organs, including the retina



FOLLOW-UP CARE

PATIENT MONITORING

- Repeated eye examinations, looking at the retina (light-sensitive lining of the back of the eye)—at 3 to 6-month intervals; confirm progressive degeneration, if the diagnosis is in doubt; will note obvious signs of degeneration over weeks in the retinas of dogs with sudden blindness due to sudden acquired retinal degeneration or SARD
- Developing and progressing cataracts (opacities in the normally clear lens)—with progressive retinal atrophy (PRA); watch for painful complications (such as glaucoma [disease of the eye, in which the pressure within the eye is increased] and inflammation of the iris and other areas in the front part of the eye [known as “uveitis”])

PREVENTIONS AND AVOIDANCE

- Do not breed animals suspected of having inherited progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time)
- Do not breed known carriers (that is, offspring of an affected animal)

POSSIBLE COMPLICATIONS

- Cataracts (opacities in the normally clear lens)
- Glaucoma (disease of the eye, in which the pressure within the eye is increased)
- Uveitis (inflammation of the iris and other areas in the front part of the eye)
- Eye trauma as a result of visual impairment
- Obesity—secondary to reduced activity

EXPECTED COURSE AND PROGNOSIS

- Inherited progressive retinal atrophy (PRA; a group of eye diseases characterized by generalized deterioration of the retina, becoming increasingly worse over time)—progresses to complete blindness; progression often slow enough for patient to adapt to visual loss; nonpainful
- Degeneration from previous inflammation or trauma—usually does not progress, unless a generalized (systemic) disease (such as toxoplasmosis) causes persistent or recurrent eye inflammation
- Sudden blindness due to sudden acquired retinal degeneration or SARD—irreversible blindness
- Transient taurine deficiency (cats)—degeneration may halt at any stage



KEY POINTS

- Patient visually impaired—condition is irreversible, but nonpainful
- Blind dogs should be watched or kept on a leash, if they are outside, not in fenced yards, or in an area with a pool
- Suggest playing with toys that make sounds
- Dogs can memorize their environment and unless the family moves or rearranges the furniture, most blind animals function well
- Apply perfume to legs of furniture to help the patient memorize the environment and identify the location of objects
- Some old blind animals with other problems (such as hearing loss or senility) may not adapt well to blindness
- Some blind animals experience behavioral changes (such as increased aggression or reduced activity)
- Animals with only one blind eye can function normally
- Blind cats may adapt better than dogs, but probably should be kept indoors

RETINAL DETACHMENT



BASICS

OVERVIEW

- “Retinal” refers to the retina; the retina is the innermost lining layer (located on the back surface) of the eyeball; it contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision
- “Retinal detachment” is the separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid”); the choroid is located immediately under the retina and is part of the middle-layer of the eyeball that contains the blood vessels

GENETICS

- Depends on the cause—dogs with hereditary opacities in the normally clear lens (known as “cataracts”) or movement of the lens out of its normal location (known as “lens luxation”) may develop separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)
- Some breeds (such as the Shih tzu, poodle, Italian greyhound) may have retinal tears or fissures and retinal detachment from primary vitreous abnormalities; the “vitreous” is the clear, gel-like material that fills the back part of the eyeball [between the lens and the retina])

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Depend on cause
- Terrier breeds—increased likelihood of developing primary lens luxation (movement of the lens out of its normal location), which may contribute to retinal tears or fissures and retinal detachment
- Breeds that develop cataracts (opacities in the normally clear lens)
- Shih tzus—appear to be susceptible to spontaneous retinal detachments, owing to abnormal vitreous; the “vitreous” is the clear, gel-like material that fills the back part of the eyeball (between the lens and the retina)

Mean Age and Range

- Depend on cause
- More common in older patients—cataracts and generalized (systemic) diseases, such as high blood pressure (known as “hypertension”, cancer, and immune-mediated disease) are often age-related

SIGNS/OBSERVED CHANGES in the ANIMAL

- Blindness or reduced vision in affected eye
- Dilated pupil with slow or no pupillary light reflex; light reflex may be near normal if detachment is sudden (acute); the “pupil” is the circular or elliptical opening in the center of the iris of the eye; light passes through the pupil to reach the back part of the eye (retina); the iris is the colored or pigmented part of the eye; the pupil constricts or enlarges (dilates) based on the amount of light entering the eye; the pupil constricts with bright light and enlarges in dim light—these actions are the “light reflexes of the pupil”
- Blood vessels or a “floating membrane” (which is the retina) may be observed easily through the pupil, just behind the lens (the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye [retina])
- Vitreous abnormalities—common; the “vitreous” is the clear, gel-like material that fills the back part of the eyeball (between the lens and the retina)
- Various changes in the appearance of the retina (light-sensitive lining of the back of the eye) may be noted when the veterinarian examines the back of the eye with an ophthalmoscope
- Other signs will depend on any underlying, generalized (systemic) diseases

CAUSES

- If the retinal detachment involves both eyes (known as “bilateral retinal detachment”), a generalized (systemic) problem is suggested
- Toxic—the individual pet is more likely to develop ill effects to a particular medication than other animals (known as “idiosyncratic reactions”), such as trimethoprim-sulfa in dogs and griseofulvin in cats

Degenerative

- End-stage progressive retinal atrophy (a group of eye diseases characterized by generalized deterioration or degeneration of the retina, becoming increasingly worse over time)—may lead to formation of holes in the retina and retinal detachment

- Long-term (chronic) glaucoma (disease of the eye, in which the pressure within the eye is increased) with stretching of the eyeball or globe and thinning of the retina

Anomalous (abnormal structure)

- Collie-eye anomaly (inherited abnormal development of the eye, leading to changes in various parts of the eye in collies); abnormal retina around the optic nerve (the nerve that runs from the back of the eye to the brain) may lead to retinal detachments
- Multiple eye abnormalities—Akitas or any breed
- Severe abnormal development of the retina (known as “retinal dysplasia”)—abnormal development of the eyes and the skeleton, characterized by dwarfism (known as “oculoskeletal dysplasia”) in Labrador retrievers, Samoyeds, English springer spaniels, and Bedlington terriers
- Abnormal development of the retinal pigment epithelium (known as “retinal pigment epithelium dysplasia”)—Australian shepherds
- Congenital (present at birth) eye defect—any young animal; congenital or juvenile retinal detachment

Metabolic

- Generalized (systemic) high blood pressure (hypertension); retinal detachment caused by high blood pressure is seen more commonly in cats than in dogs
- Inadequate levels of thyroid hormone (known as “hypothyroidism”)
- Increased protein in the blood leading to sludging of the blood (known as “hyperviscosity”)
- Increased packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood); hemoglobin concentration (hemoglobin is the compound in the red-blood cells that carries oxygen to the tissues of the body); and red-blood cell (RBC) count above the normal ranges (known as “polycythemia”)
- Low levels of oxygen in the tissues (hypoxia), with bleeding complicationsDogs—generalized (systemic) high blood pressure (hypertension) due to any cause, such as kidney failure, low levels of thyroid hormone (hypothyroidism), high levels of cholesterol in the blood (known as “hypercholesterolemia”)
- Cats—most often caused by generalized (systemic) high blood pressure (hypertension) either as a primary condition or secondary to kidney failure or excessive levels of thyroid hormone (known as “hyperthyroidism”)

Cancer or Tumors

- Any primary cancer or one that has spread to other body tissues (known as “metastatic cancer”)
- Commonly associated with the following cancers: multiple myeloma, lymphoma, and cancer of the eye (ciliary body adenocarcinoma or melanoma)
- High blood pressure (hypertension) secondary to adrenal gland tumors, like pheochromocytoma (dogs and cats), rare

Infectious

- Infectious inflammation of the retina (known as “retinitis”) or inflammation of the choroid and retina (known as “chorioretinitis”); the “choroid” is located immediately under the retina and is part of the middle-layer of the eyeball that contains the blood vessels—may cause localized or more widespread retinal detachment
- Infection may extend from or to the central nervous system (brain)

Immune-mediated/Inflammatory

- Immune-complex disease—may cause inflammation of the blood vessels (known as “vasculitis”) or inflammation of eye tissues that may result in retinal detachment
- Dogs—systemic lupus erythematosus (autoimmune disease in which body attacks its own skin and possibly other organs) or uveodermatologic syndrome (a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris [anterior uveitis] and coexistent inflammation of the skin [dermatitis], characterized by loss of pigment in the skin of the nose and lips)
- Cats—periarteritis nodosa (immune-mediated disease leading to inflammation of small and medium-sized arteries); systemic lupus erythematosus (autoimmune disease in which body attacks its own skin and possibly other organs)

Unknown Cause (Known as “Idiopathic Disease”)

- If all other causes are ruled out, including tears or fissures of the retina
- Steroid-responsive retinal detachment of unknown cause (condition known as “idiopathic steroid-responsive detachment”)—reported in giant-breed dogs, but may occur in any breed

Trauma

- Affecting both eyes (bilateral)—probably never occurs
- Penetrating injury or foreign body that causes tears or fissures of the retina or bleeding within the eye—may cause partial or complete retinal detachment
- Severe blunt trauma with inflammation or bleeding
- Surgical trauma—may contribute to tears or fissures of the retina

RISK FACTORS

- Generalized (systemic) high blood pressure (hypertension)
- Old age
- Opaque lens that shrinks in size and then liquifies (known as a “hyperimmune cataract”)

- Movement of the lens out of its normal location (known as “lens luxation”)
- Surgical removal of the lens; the “lens” is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye (retina)



TREATMENT

HEALTH CARE

- Depends on the physical condition of the patient
- Usually outpatient
- Sudden (acute) blindness—vision may be restored if the underlying cause of the separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment) is identified and treated rapidly; make every attempt to determine the cause of retinal detachment
- Degeneration of retinal tissue occurs rapidly following separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)—provide therapy, whether surgical or medical, as soon as possible after diagnosis
- A veterinary ophthalmologist (eye specialist) may be able to provide surgical treatment/reattachment for some types of retinal detachment
- Treatment as needed for any associated generalized (systemic) disease

ACTIVITY

- Restrict until retinal reattachment has occurred (if reattachment is possible)
- Supervise irreversibly blind patients

SURGERY

- Some types of retinal detachment may be repaired surgically; refer patient to a veterinary ophthalmologist (eye specialist)
- Procedure to repair a detached retina and hold it in place using a laser (known as “laser retinopexy”)—may reverse some retinal detachments associated with collie-eye anomaly (inherited abnormal development of the eye, leading to changes in various parts of the eye in collies); may stabilize partial/small retinal detachments



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Depend on underlying generalized (systemic) causes, which should be identified and treated appropriately
- Steroids administered by mouth or injection, such as prednisone—if generalized fungal infection (known as “systemic mycosis”) is ruled out as a cause of the retinal detachment and if the detachment is believed to be immune-mediated; may help retinal reattachment; for immune-mediated disease, taper medications very slowly over months, as directed by your pet’s veterinarian
- Anti-inflammatory doses of prednisone—may be useful for retinal detachments of an infectious nature, as long as the underlying disease is being treated
- Medications to decrease blood pressure (known as “antihypertensive agents”)—amlodipine; others (such as propranolol) can be used if amlodipine fails to control high blood pressure (hypertension)
- Angiotensin-converting enzyme (ACE) inhibitors (such as enalapril or benazepril) can be used to treat cats with high blood pressure (hypertension) that is not responsive to amlodipine alone; may be important in cats that have kidney failure and protein in their urine (known as “proteinuria”)
- Chemotherapy—suggested for treatment of cancer (such as lymphoma or multiple myeloma)
- Azathioprine (dogs)—type of chemotherapy, used to control inflammation; may be required in addition to steroids for uveodermatologic syndrome (a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris [anterior uveitis] and coexistent inflammation of the skin [dermatitis], characterized by loss of pigment in the skin of the nose and lips) or immune-mediated retinal detachment of unknown cause (idiopathic immune-mediated detachment); avoid use in cats



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on underlying cause and type of medical treatment
- Azathioprine—an initial complete blood count (CBC) and then a follow-up CBC should be obtained every 1 to 2 weeks for the first 1 to 3 months of treatment; monitor every 1 to 2 months for bone-marrow suppression, leading to low red-blood cell and low white-blood cell counts (if noted, the dose should be reduced or treatment discontinued); in addition, initial and follow-up blood work should be performed to evaluate for liver or pancreas toxicity
- Monitor blood pressure in cases with high blood pressure (hypertensive animals)

POSSIBLE COMPLICATIONS

- Permanent blindness
- Cataracts (opacities in the normally clear lens)
- Glaucoma (increased pressure within the eye)
- Long-term (chronic) eye pain
- Death, if retinal detachment is secondary to a generalized (systemic) disease process

EXPECTED COURSE AND PROGNOSIS

- Prognosis for vision with complete retinal detachment—guarded; the exception is a disease of the retina due to high blood pressure (known as “hypertensive retinopathy”), which is diagnosed and treated promptly
- Blindness—may develop in days to weeks, even if retinal reattachment occurs
- Vision may return, if the underlying cause is removed and reattachment occurs
- Localized or multiple areas of inflammation of the choroid and retina (chorioretinitis)—does not impair vision markedly; will leave scars; the “choroid” is located immediately under the retina and is part of the middle-layer of the eyeball that contains the blood vessels
- Generalized (systemic) disease or cancer with eye involvement—may influence the prognosis for life



KEY POINTS

- Retinal detachment (especially if both eyes are involved [bilateral disease]) may be a sign of generalized (systemic) disease, so diagnostic testing is important
- Retinal detachment associated with movement of the lens out of its normal location (lens luxation) or cataract surgery has a potential to affect both eyes, so both eyes should be observed closely
- Retinal detachments may be reversible, with return of vision, if the underlying cause is treated and the detachment is caught early
- Blind pets, especially cats, can adapt remarkably well and live a good quality life

RETINAL HEMMORHAGE (BLEEDING INTO THE RETINA)



BASICS

OVERVIEW

- “Retinal” refers to the retina; the retina is the innermost lining layer (located on the back surface) of the eyeball; it contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision
- “Retinal hemorrhage” is local or generalized areas of bleeding into part or all layers of the retina
- May be sudden (acute) or long-term (chronic) bleeding
- “Retinal detachment” is the separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid”), which can lead to bleeding into the retina; the “choroid” is located immediately under the retina and is part of the middle-layer of the eyeball that contains the blood vessels

GENETICS

- Cause may have a genetic basis and be highly breed and age specific—young collies with collie-eye anomaly (inherited abnormal development of the eye, leading to changes in various parts of the eye in collies); Labrador retrievers with congenital (present at birth) abnormal development of the vitreous and retina (known as “vitreoretinal dysplasia”)—the “vitreous” is the clear, gel-like material that fills the back part of the eyeball
- Hereditary, breed-specific, congenital (present at birth) defects that might cause separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment) or severe abnormal development of retina (known as severe “retinal dysplasia”)—collies and Sheltland sheepdogs with collie-eye anomaly (inherited abnormal development of the eye, leading to changes in various parts of the eye); Australian shepherds with defective eye development related to the merle color pattern (known as “merle ocular dysgenesis”); Labrador retrievers, Sealyham terriers, Bedlington terriers, and English springer spaniels with abnormal development of the retina (retinal dysplasia); and miniature schnauzers with retinal dysplasia and embryonic developmental abnormalities of the eye (known as “persistent hyperplastic primary vitreous,” the “vitreous” is the clear, gel-like material that fills the back part of the eyeball [between the lens and the retina])

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Generalized (systemic) high blood pressure (known as “hypertension”) seen in older cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on underlying cause
- Often no signs are observed
- Vision loss
- Bumping into objects
- Evidence of bleeding elsewhere—small, pinpoint areas of bleeding (known as “petechia”); bruises or purplish patches under the skin, due to bleeding (known as “ecchymoses”); black, tarry stools due to the presence of digested blood (known as “melena”); blood in the urine (known as “hematuria”)
- Whitish appearing pupil (known as “leukocoria”), with or without reddish coloration behind the lens
- Abnormal light reflexes of the pupil; the pupil is the circular or elliptical opening in the center of the iris of the eye; light passes through the pupil to reach the back part of the eye (retina); the iris is the colored or pigmented part of the eye; the pupil constricts or enlarges (dilates) based on the amount of light entering the eye; the pupil constricts with bright light and enlarges in dim light—these actions are the “light reflexes of the pupil”

CAUSES

Congenital (present at birth)

- Separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment) secondary to congenital (present at birth) malformation of the retina
- Abnormal development of the vitreous and retina (vitreoretinal dysplasia) or the retina (retinal dysplasia); the “vitreous” is the clear, gel-like material that fills the back part of the eyeball (between the lens and the retina)

Acquired (condition that develops sometime later in life/after birth)

- Trauma

- Generalized (systemic) high blood pressure (hypertension), especially in old cats—kidney disease; heart disease; increased levels of thyroid hormone (known as “hyperthyroidism”); increased levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); high blood pressure of unknown cause (condition known as “idiopathic hypertension”)

- Intoxication—various compounds, including dicumarol (an anticoagulant, to stop blood from clotting); paracetamol (a pain reliever); sulfonamide (type of antibiotic)

- Infectious disease—*Rickettsia rickettsia* (Rocky Mountain spotted fever), *Ehrlichia*, cryptococcosis (a deep fungal infection)

- Cancer—such as lymphoma, plasma-cell myeloma

- Blood disorders—blood-clotting disorders (such as von Willebrand’s disease); severely low red-blood cell counts (known as “severe anemia”); low platelet count (known as “thrombocytopenia”); immune protein from a single clone of cells in the gamma region of a protein analysis of blood (known as a “monoclonal gammopathy”) and a condition in which increased protein in the blood leads to sludging of the blood (known as “hyperviscosity syndrome”)

- Retinal disease secondary to diabetes mellitus (“sugar diabetes,” condition known as “diabetic retinopathy”)

- Separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)

- Immune-mediated inflammation of blood vessels (known as “vasculitis”)

RISK FACTORS

- Generalized (systemic) high blood pressure (hypertension)

- Blood disorders



TREATMENT

HEALTH CARE

- Usually initially treated as inpatients—sometimes in intensive care for maximal follow-up

- Intoxications—often require specific treatment

- Consider referral to a veterinary ophthalmologist (eye specialist) for a more detailed examination

ACTIVITY

- Separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)—cage rest until the retina is reattached (if reattachment is possible)

SURGERY

- Surgery—refer patient to a veterinary ophthalmologist (eye specialist) for surgical removal of the vitreous (known as “vitrectomy”) and/or retinal reattachment surgery; the “vitreous” is the clear, gel-like material that fills the back part of the eyeball (between the lens and the retina)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Depend on underlying cause

- Steroids administered by mouth or by injection—if workup is declined and infectious disease is unlikely; prednisolone; especially for separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment) as a sequela to trauma

- Chloramphenicol or other broad-spectrum antibiotic administered by mouth or by injection—if bacterial infectious disease is suspected; may be administered at the same time as steroids are being administered

- Primary generalized (systemic) high blood pressure (hypertension)—treat as required; often combined with steroid treatment, medications to remove excess fluid from the body (known as “diuretics,” such as furosemide), and calcium channel blockers

- Azathioprine administered by mouth—type of chemotherapy, used to control inflammation for immune-mediated separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment); combine with steroid treatment; may be used in immune-mediated disease involving the back of the eye

- Itraconazole (an anti-fungal medication)—for cryptococcosis or other deep fungal infection
- Flunixin meglumine—may be used in dogs if eye is inflamed and infectious causes have not been ruled out



FOLLOW-UP CARE

PATIENT MONITORING

- Repeated monitoring—required to ensure that condition subsides and to follow changes in the retina
- Azathioprine treatment—a complete blood count (CBC), platelet count, and liver enzyme analysis should be performed every 2 weeks for the first 2 months, then periodically

POSSIBLE COMPLICATIONS

- Separation of the back part of the eye (retina) from the underlying, vascular part of the eyeball (retinal detachment)
- Blindness
- Impaired vision
- Long-term (chronic) inflammation of the iris and other areas in the front part of the eye (known as “uveitis”)
- Glaucoma (increased pressure within the eye)

EXPECTED COURSE AND PROGNOSIS

- Depend on cause
- Blood from preretinal bleeding—usually reabsorbed within a few weeks to several months, if localized
- Larger or repeated bleeding—may be followed by development of scar tissue, which may cause retinal detachment
- Bleeding within the retina—blood is reabsorbed within several weeks to months; may produce retinal scarring



KEY POINTS

- Blind pets, especially cats, can adapt remarkably well and live a good quality life
- Consider euthanasia of young puppies with severe bleeding in both eyes (known as “bilateral hemorrhage”) due to congenital (present at birth) abnormalities
- Dogs with bleeding in one eye due to congenital (present at birth) abnormalities can function as pets, but should not be used for breeding

RHINITIS AND SINUSITIS (INFLAMMATION OF THE NOSE AND SINUSES)



BASICS

OVERVIEW

- Rhinitis—inflammation of the lining of the nose
- Sinusitis—inflammation and irritation of the sinuses
- The nasal cavity communicates directly with the sinuses; thus inflammation of the nose (rhinitis) and inflammation of the sinuses (sinusitis) often occur together (known as “rhinosinusitis”)
- “Upper respiratory tract” (also known as the “upper airways”) includes the nose, nasal passages, throat (pharynx), and windpipe (trachea)
- “Lower respiratory tract” (also known as the “lower airways”) includes the bronchi, bronchioles, and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Short-nosed, flat-faced (known as “brachycephalic”) cats are more prone to long-term (chronic) inflammation of the nose (rhinitis), and possibly fungal rhinitis
- Dogs with a long head and nose (known as “dolichocephalic dogs,” such as the collie and Afghan hound) are more prone to *Aspergillus* (a type of fungus) infection and nasal tumors

Mean Age and Range

- Cats—sudden (acute) viral inflammation of the nose and sinuses (rhinosinusitis) is more common in young kittens (6 to 12 weeks of age) or unvaccinated cats; red masses in the nasal cavity (known as “inflammatory polyps”) are more common in young cats
- Congenital (present at birth) diseases (such as cleft palate) are more common in young animals
- Tumors/cancer and dental disease—are more common in older animals
- Foreign bodies are more common in young dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sneezing, discharge from the nose, bleeding in the nose and nasal passages (known as “epistaxis” or a “nosebleed”)
- Discharge—clear initially, then it may contain mucus and/or pus; it may be blood tinged or may contain blood
- Discharge from one nostril suggests the presence of a foreign body, tooth-root abscess, tumor/cancer, or fungal infection; inflammation of the nose for unknown reason (so called “idiopathic inflammatory rhinitis”) also may present with discharge from only one nostril
- Discharge from both nostrils is more common with viral or bacterial inflammation of the nose and sinuses (rhinosinusitis), disease involving the throat (known as “pharyngeal disease”), or congenital (present at birth) abnormalities
- Facial deformity—usually associated with fungal disease or tumors/cancer
- Reverse sneezing is more common in affected dogs than in cats; “reverse sneezing” is a sudden attack or spasm of noisy intake of air (inspiration) to clear accumulated discharge from the back of the nasal passages into the throat, from which it is swallowed; reverse sneezing is a response to irritation at the back of the nasal passages
- Lack of appetite is more common in affected cats than in dogs
- Decreased air flow through the nasal passages suggests tumors/cancer or fungal infection by *Cryptococcus*
- May have abnormalities in the mouth (such as a tooth-root abscess, abnormal opening between the mouth and nose [known as an “oronasal fistula”], or ulcers)
- Increased sensitivity to touching the windpipe or trachea or cough is possible
- Excessive tears or overflow of tears (known as “epiphora”), inflammation of the moist tissues of the eye (known as “conjunctivitis”), and/or Horner’s syndrome (condition in which one pupil is small or constricted, the eyelid droops, and the eyeball is withdrawn into the socket), indicating middle-ear disease, may be present
- Loss of pigment in the skin (known as “depigmentation”) of the nose suggests aspergillosis, a fungal disease
- Abnormalities involving the back of the eye (the retina) may be seen, associated with fungal inflammation of the nose (rhinitis)
- Nervous system signs may be seen and suggest breach of the cribriform plate (bony plate located between the nasal passages and the

brain) by fungal disease or tumors/cancer

- Lymph nodes may be enlarged due to infectious or inflammatory disease or to cancer

CAUSES

Dogs

Primary Inciting Causes

- Fungal disease—*Aspergillus fumigatus* most common; *Penicillium*, *Rhinosporidium*, *Blastomycoses*, *Cryptococcus* are rare causes
- Tooth-root abscess
- Foreign body
- Congenital (present at birth) abnormalities (such as cleft palate)
- Parasitic causes—nasal mites (*Pneumonyssoides caninum*), nasal worm of dogs (*Eucoleus boehmi*)
- Cancer in the nose—adenocarcinoma most common; chondrosarcoma, osteosarcoma, or lymphoma also seen
- Immune-mediated inflammation of the nose (rhinitis)—allergic rhinitis is rare; inflammation of the nose of unknown cause, characterized by the presence of lymphocytes and plasma cells (so called “idiopathic lymphoplasmacytic rhinitis”) is more common; “lymphocytes” are a type of white-blood cell that are formed in lymphatic tissues throughout the body—lymphocytes are involved in the immune process; “plasma cells” are specialized white-blood cells—plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease
- Other infectious diseases include canine distemper or *Bordetella bronchiseptica* (one cause of kennel cough)
- Local trauma may cause bone deformity and increase the likelihood of developing long-term (chronic) inflammation of the nose (rhinitis)

Secondary Causes

- Lower airway disease (bronchopneumonia) may cause signs of inflammation of the nose (rhinitis)
- Bleeding from the nose or nasal passages (epistaxis or nose bleed) can be related to high blood pressure (hypertension), decreased number of platelets in the blood (known as “thrombocytopenia”), a disorder that leads to dysfunction of the platelets (known as “thrombocytopathia”), or rarely other blood-clotting disorders (known as “coagulopathies”); “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- Dogs with vomiting can aspirate into the nasopharynx (the part of the throat that communicates with the nasal cavity)

Cats

Primary Inciting Causes

- Viral infections—feline herpesvirus-1 and calicivirus cause 90% of sudden (acute) infections and/or *Chlamydophila* (a type of bacteria) inflammation of the nose and sinuses (rhinosinusitis)
- *Bordetella bronchiseptica* can be a primary disease-causing agent in cats, but number of cases is uncertain
- Feline immunodeficiency virus (FIV) and feline leukemia virus (FeLV) infections or bacterial co-infections are common in long-term (chronic) inflammation of the nose (rhinitis)
- Fungal disease—*Cryptococcus* most common; also consider *Aspergillus* and *Penicillium* (rare in cats)
- Cancer—adenocarcinoma and lymphoma most common
- Inflammatory masses that develop from the middle ear or eustachian tube (known as “nasopharyngeal polyps”) in young cats
- Tooth-root abscess
- Allergic inflammation of the nose (rhinitis) is rare
- Foreign body
- Parasitic infection
- Congenital (present at birth) abnormalities (such as cleft palate)

Secondary Causes

- Bleeding from the nose or nasal passages (epistaxis or nose bleed) related to a blood-clotting disorder (coagulopathy) or high blood pressure (hypertension) is less common in cats than in dogs
- Cats with vomiting can aspirate into the nasopharynx (the part of the throat that communicates with the nasal cavity)

RISK FACTORS

- Short-nosed, flat-faced (brachycephalic) cats—inflammation of the nose and sinuses (rhinosinusitis)
- Dogs with a long head and nose (dolichocephalic dogs, such as the collie and Afghan hound)—fungal disease



TREATMENT

HEALTH CARE

- Depends on the underlying cause
- Humidification to moisten the nasal passages; saline infusion into the nasal passages is helpful, if tolerated by the animal
- Clean discharges from the nostrils and area around the nose

ACTIVITY

- No change unless in breathing distress

DIET

- Soft or warmed food to increase appetite

SURGERY

- Surgery may be necessary to obtain a biopsy or to remove a foreign body or mass
- Decreasing the size of a tumor (known as “surgical debulking”) may improve survival time of the animal, if combined with radiation therapy
- Useful for removal of polyps



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Antibiotics

- Potential bacterial disease-causing agents are isolated more commonly in cats with inflammation of the nose and sinuses (rhinosinusitis) than in healthy cats
- Antibiotic therapy should be based on bacterial culture and sensitivity testing
- May help with secondary bacterial inflammation of the nose (rhinitis) in dogs, however, antibiotics will not resolve the underlying primary problem
- Tetracycline—helpful with *Chlamydophila* inflammation of the nose (rhinitis); may need long-term doxycycline therapy for 6 to 8 weeks
- Chloramphenicol also is effective against *Chlamydophila*

Antifungal Medications

- To treat cryptococcosis or aspergillosis

Human alpha-interferon

- Anecdotal at this point

L-lysine

- Decreases feline herpesvirus-1 replication in cell culture and virus shedding; may be useful in long-term (chronic) herpesvirus infection

Anti-Inflammatory Agents

- Nonsteroidal anti-inflammatory drugs (NSAIDs), such as piroxicam (used for treatment of nasal tumors, either as sole agent or in conjunction with chemotherapy)
- Steroids—prednisolone for allergic inflammation of the nose (allergic rhinitis); anti-inflammatory doses for cats with long-term (chronic) inflammation of the nose and sinuses (rhinosinusitis) or lymphoplasmacytic inflammation of the nose (rhinitis) in dogs

Antihistamines

- Efficacy is debated—clemastine or hydroxyzine

Anti-Parasitic Medications for Nasal Mites

- Ivermectin administered by mouth, once weekly for 3 to 4 treatments or milbemycin administered by mouth once weekly for 3 weeks



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on the underlying cause
- Clinical assessment and monitoring for relapse

PREVENTIONS AND AVOIDANCE

- Depend on the underlying cause
- Vaccinations in kittens can lessen severity and duration of feline herpesvirus-1 or calicivirus infection
- Consider removing chronically affected cats from catteries; use lysine to decrease feline herpesvirus-1 shedding

POSSIBLE COMPLICATIONS

- Depend on the underlying cause and extent of disease
- Fungal or tumor invasion into the brain (through the cribriform plate, the bony plate located between the nasal passages and the brain)

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause and extent of disease
- Sudden (acute) viral/bacterial inflammation of the nose (rhinitis)—carries good prognosis
- Long-term (chronic) inflammation of the nose (rhinitis) is frustrating to the owners and veterinarians
- Fungal disease—fair to guarded prognosis, depending on invasiveness of the fungal infection
- Cancer—3 to 5 months' survival time, with no treatment; life expectancy can be extended up to 20 to 23 months with radiation therapy



KEY POINTS

- Signs of long-term (chronic) inflammation of the nose (rhinitis) in dogs and cats can be controlled, but rarely are eliminated

ROCKY MOUNTAIN SPOTTED FEVER



BASICS

OVERVIEW

- A tick-borne rickettsial disease, caused by *Rickettsia rickettsii*, that affects dogs and is considered the most important rickettsial disease in people
- Antibodies to *Rickettsia akari* (causative agent of rickettsial pox in people) have been found in dogs from New York, NY; unknown if this *Rickettsia* causes disease in dogs
- Other as yet undefined rickettsial organisms may cause clinical signs in dogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Purebred dogs seem more prone to developing clinical illness than do mixed-breed dogs
- German shepherd dogs—more common

Mean Age and Range

- Any age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Fever—within 2 to 3 days of attachment of a tick carrying *Rickettsia rickettsii*
- Sluggishness (lethargy)
- Depression
- Lack of appetite (known as “anorexia”)
- Swelling and fluid build-up in the tissues (known as “edema”)—face, lips, scrotum, prepuce, ears, legs
- Stiff gait
- Spontaneous bleeding—sneezing; bleeding in the nose and nasal passages (known as “epistaxis” or a “nosebleed”); black, tarry stools due to the presence of digested blood (known as “melenia”); blood in the urine (known as “hematuria”)
- Nervous system signs—wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”); head tilt; altered mental status; seizures
- Eye pain
- Ticks may be present on the dog
- May have death of tissues (known as “necrosis”) on the legs
- Inflammation of the moist tissues of the eyes (known as “conjunctivitis”)
- Difficulty breathing (known as “dyspnea”), exercise intolerance, breathing distress, increased lung sounds when listening to the chest with a stethoscope
- Generalized enlarged lymph nodes (known as “lymphadenopathy”)
- Muscular pain (known as “myalgia”) and/or joint pain (known as “arthralgia”)
- Small, pinpoint areas of bleeding (known as “petechia”)
- Bruises or purplish patches, due to bleeding (known as “ecchymoses”)—involving the eyes, mouth, and genital regions; seen in 20% of patients
- Irregular heart beats (known as “cardiac arrhythmias”)—sudden death
- Blood-clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”) and death from shock—in severe, sudden (acute) cases

CAUSES

- *Rickettsia rickettsii*

RISK FACTORS

- Exposure to ticks carrying *Rickettsia rickettsii*
- Co-infection with other tick borne disease-causing agents



TREATMENT

HEALTH CARE

- Inpatient, until stable and showing response to treatment
- Dehydration—balanced fluids, administered cautiously to avoid increasing fluid build-up in the tissues (such as the brain [known as “cerebral edema”] or lungs [known as “pulmonary edema”])
- Low red-blood cell count (known as “anemia”)—blood transfusion
- Bleeding from low platelet count (known as “thrombocytopenia”)—platelet-rich plasma or a blood transfusion; “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding

ACTIVITY

- Restricted

SURGERY

- If surgery is required for other reasons, blood transfusion may be needed to correct low red-blood cell counts (anemia) and/or low platelet counts (thrombocytopenia)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Doxycycline—synthetic derivative of tetracycline, administered by mouth for 10 days or intravenously (IV) for 5 days, if patient is vomiting
- Prednisone—used to decrease inflammation; may be given early in course of disease
- Other antibiotics (tetracycline, chloramphenicol, and enrofloxacin)—are effective, if used early



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor platelet count every 3 days until it has returned to normal

PREVENTIONS AND AVOIDANCE

- Control tick infestation on dogs—use dips or sprays containing dichlorvos, chlorfenvinphos, dioxathion, propoxur, or carbaryl; any product used to control ticks should be used only as directed by the product label
- Flea and tick collars—may reduce re-infestation of ticks; reliability has not been proven
- Avoid tick-infested areas
- Environment—tick eradication impossible; tick populations are maintained in rodents and other reservoir hosts
- Removing ticks by hand—use gloves; ensure mouth parts are removed, because a foreign body reaction is likely to result in the skin, if they are left in place

POSSIBLE COMPLICATIONS

- Death

EXPECTED COURSE AND PROGNOSIS

- Early antibiotic treatment—reduces fever and improves patient’s attitude within 24 to 48 hours
- Platelet counts—should return to normal within 2 to 4 days after initiating treatment

- Serologic tests (blood tests that detect the presence of antibodies to a certain disease-causing agent or antigen; an “antibody” is a protein that is produced by the immune system in response to a specific antigen)—lower in treated than in untreated dogs; titers remain positive during the recovery period
- Naturally infected dogs never seem to become reinfected
- Clinical Rocky Mountain spotted fever—variable in severity of disease; lasts 2 to 4 weeks, if untreated
- Sudden (acute) cases—excellent prognosis with appropriate treatment
- Central nervous system disease—poor prognosis



KEY POINTS

- Prognosis—good in sudden (acute) cases, with appropriate and prompt treatment
- Response occurs within hours of treatment
- If treatment is not instituted until central nervous system signs occur or later in the disease process, death rate is high; patient with central nervous system signs may die within hours

DISEASE CAUSED BY *SALMONELLA*, A TYPE OF BACTERIA (SALMONELLOSIS)



BASICS

OVERVIEW

- “Salmonellosis” is a bacterial disease caused by many different serotypes of *Salmonella*; “serotypes” are subdivisions of a species that are different from other strains
- *Salmonella* infection may cause inflammation of the intestines (known as “enteritis”), generalized disease secondary to spread of bacteria in the blood (known as “septicemia” or “blood poisoning”), and abortions

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Dogs—clinical disease is seen in newborn or immature puppies and in pregnant female dogs (known as “bitches”); most adult carrier dogs clinically are normal; a “carrier state” is one in which the animal has no signs of disease, but harbors *Salmonella* and can transmit it to other animals
- Cats—adults are highly resistant

SIGNS/OBSERVED CHANGES in the ANIMAL

- Disease severity varies; animal may be infected, but has no signs of disease (known as a “subclinical infection”) leading to a carrier state, in which the animal harbors *Salmonella*, sheds it in bowel movement and thus transmits it to other animals; mild, moderate, and severe clinical cases may be seen in newborns and stressed adult dogs and cats
- Subclinical infection more common than clinical disease, which is rare
- Carrier animals—no clinical signs
- Diarrhea with mucus and/or blood
- Vomiting
- Dehydration
- Fever
- General signs of discomfort and “not feeling well” (known as “malaise”)
- Lack of appetite (known as “anorexia”)
- Sluggishness (lethargy), depression
- Abdominal pain
- Weight loss
- Vaginal discharge/abortion—dogs
- Animals with the presence of bacteria in the blood (known as “bacteremia”) and generalized disease caused by the spread of bacteria in the blood (septicemia or blood poisoning); shock associated with generalized bacterial infection (sepsis; condition known as “septic shock”); or the presence of bacterial toxins in the blood [known as “endotoxemia”]—pale gums and moist tissues of the body (known as “mucous membranes”); weakness; circulatory collapse; rapid heart rate (known as “tachycardia”); rapid breathing (known as “tachypnea”)
- Localized infections outside of the intestinal tract—inflammation of the moist tissues of the eye (known as “conjunctivitis”); uterus/abortion; inflammation of the tissues under the skin that tends to spread (known as “cellulitis”); infection/inflammation of the lining of the chest, leading to accumulation of pus in the space between the chest wall and the lungs (condition known as “pyothorax”)
- Cats—may exhibit syndrome of a long-term (chronic) illness (without gastrointestinal signs) characterized by persistent fever; prolonged illness with vague, nonspecific clinical signs; and abnormal white-blood cell count
- Recovering patients—may exhibit long-term (chronic) intermittent diarrhea for 3 to 4 weeks; may shed *Salmonella* in bowel movement for 6 weeks or longer

CAUSES

- Any one of more than 2000 serotypes of *Salmonella*; “serotypes” are subdivisions of species that are different from other strains
- Two or more simultaneous serotypes in a host animal are not uncommon

RISK FACTORS

Disease Agent

- *Salmonella* serotype—variable disease-causing factors, infectious dose, and route of exposure
- Host factors that increase likelihood of developing clinical signs
- Age—newborn or young dogs and cats; immature immune system
- Overall health status—debilitated young animals or adults: other coexistent disease, presence of parasites; young animals: immature gastrointestinal tract, poorly developed normal intestinal bacteria
- Disrupted gastrointestinal bacteria flora (adult cats)—antibiotic treatment; subsequent exposure to *Salmonella* during hospitalization

Environmental Factors

- Eating of feces or bowel movement (known as “coprophagia”)—spreads infection
- Dehydrated pet food—may harbor *Salmonella*; kibble and dog biscuits usually not as risky
- Pig-ear dog treats contaminated by *Salmonella*
- Horse meat fed to exotic cats
- Grooming habits—may result in *Salmonella*-contaminated hair coat, which contaminates cage or run environment, feed and water dishes
- Dense population of animals—kennels/boarding facilities, shelters, catteries; overcrowded housing; unsanitary conditions; exposure to other infected (or carrier) animals—build-up of *Salmonella* in the environment; increased opportunity for exposure to *Salmonella*; stress factors

Hunting/Stray Animals

- Scavenging for food—exposure to garbage, contaminated food/water, dead animals
- Exposure to other infected (or carrier) animals
- Exposure to infected raw meat

Hospitalized Animals

- Exposure of hospitalized animal to *Salmonella* during a period of stress or activation (by stress) of pre-existing carrier *Salmonella* infection, especially in animals treated with antibiotics

Vaccinations

- Death in kittens (likely to be infected by *Salmonella*, but not showing signs [subclinical infection]) following vaccination with some types of panleukopenia vaccine



TREATMENT

HEALTH CARE

- Outpatient—uncomplicated intestinal disease (without the presence of bacteria in the blood [bacteremia]) and carrier states; a “carrier state” is one in which the animal has no signs of disease, but harbors *Salmonella* and can transmit it to other animals
- Inpatient—with presence of bacteria in the blood (bacteremia) and/or generalized disease caused by the spread of bacteria in the blood (septicemia or blood poisoning) and for inflammation of the stomach and intestines (known as “gastroenteritis”) in newborns and immature animals that are debilitated rapidly by diarrhea
- Varies according to severity of illness—assess dehydration, body weight, ongoing fluid loss, shock, packed cell volume (“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood) and total protein (a quick laboratory test that provides general information on the level of protein in the fluid portion of the blood), electrolytes, acid–base status

Uncomplicated Inflammation of the Stomach and Intestines (Gastroenteritis)

- Supportive care—fluid and electrolyte replacement
- Balanced fluids (such as lactated Ringer’s solution), administered intravenously
- Oral fluids—special glucose solutions; for secretory diarrhea
- Plasma transfusions—if serum albumin (a protein in the blood) is less than 2 g/dl

Newborns, Aged, and Debilitated Animals

- Plasma transfusions
- Supportive care—as for Uncomplicated Gastroenteritis

ACTIVITY

- Isolate inpatients—all patients in sudden (acute) stage of disease may shed a large number of *Salmonella* in their bowel movement
- Restrict activity with cage rest, monitor, and provide warmth—suddenly (acutely) ill patients; animals with bacteria in their blood

(bacteremia) or generalized disease caused by the spread of bacteria in the blood (septicemia or blood poisoning); and animals that have been ill for a long time (chronically ill animals)

DIET

- Restrict food for 24 to 48 hours; gradually introduce a highly digestible, low-fat diet



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Carrier State (the animal has no signs of disease, but harbors *Salmonella* and can transmit it to other animals)

- Antibiotics—should not be administered
- Quinolone drugs—demonstrated clearing of carrier states in people; more controlled trials in animals needed

Uncomplicated Inflammation of the Stomach and Intestines (Gastroenteritis)

- Antibiotics not indicated
- Locally acting medications to protect the lining of the intestines

Newborns, Aged, and Debilitated Animals

- Steroids—shown to reduce death rate in animals with shock due to the presence of bacterial toxins in the blood (known as “endotoxic shock”)
- Antibiotics—should be administered; ideally, perform bacterial culture and sensitivity testing to determine appropriate antibiotic; examples include trimethoprim-sulfa, enrofloxacin, norfloxacin, and chloramphenicol



FOLLOW-UP CARE

PATIENT MONITORING

- Fecal bacterial culture to check for *Salmonella* in the bowel movement—repeat monthly for few months to assess development of carrier state
- Other animals—monitor for secondary spread of infection
- Contact your pet’s veterinarian, if patient shows signs of recurring disease

PREVENTIONS AND AVOIDANCE

- Keep animals healthy—proper nutrition; no raw meat; vaccinate for other infectious diseases; clean and disinfect cages, runs, and food and water dishes frequently; store food and feeding utensils properly
- Reduce overcrowding—kennels/boarding facilities, shelters, and catteries
- New arrivals—isolate and screen; monitor for sickness before introducing them to other animals
- Experimental live attenuated vaccine shows promise, especially for racing dogs
- Important to protect animals being treated with antibiotics from exposure to a *Salmonella*-contaminated environment

POSSIBLE COMPLICATIONS

- Spread of infection within the household to other animals or to people is not uncommon
- Development of long-term (chronic) infection with diarrhea
- Recurrence of disease with stress
- Death

EXPECTED COURSE AND PROGNOSIS

- Uncomplicated inflammation of the stomach and intestines (gastroenteritis)—prognosis excellent; frequently self-limited; patients recover with good nursing care
- Recovered animals may shed *Salmonella* intermittently for months or longer as a recovered carrier (the animal has no signs of disease, but harbors *Salmonella* and can transmit it to other animals)
- Newborn, aged, stressed animals—can develop generalized disease caused by the spread of bacteria in the blood (septicemia or blood poisoning); can be severe and debilitating; may lead to death, if untreated



KEY POINTS

- People should wash hands frequently
- Restrict access to the pet in sudden (acute) stages of the disease
- A large number of *Salmonella* may be shed in the bowel movement; recovered animals may shed *Salmonella* intermittently for months or longer as a recovered carrier
- A “carrier state” is one in which the animal has no signs of disease, but harbors *Salmonella* and can transmit it to other animals or people

SEIZURES (CONVULSIONS, STATUS EPILEPTICUS) IN CATS



BASICS

OVERVIEW

- “Seizures” are periods of uncontrolled electrical activity in the brain (also known as “convulsions”); “status epilepticus” is repeated or prolonged seizure activity
- “Epilepsy”—disorder characterized by recurring seizures that originate from the brain
- “Idiopathic epilepsy”—epilepsy of unknown cause; syndrome that involves only epilepsy, with no demonstrable underlying brain lesion or other nervous system signs; rare in cats
- “Symptomatic epilepsy”—syndrome in which epileptic seizures are the result of identifiable, structural brain lesions; frequent in cats
- “Probably symptomatic epilepsy”—when symptomatic epilepsy is suspected, but a lesion cannot be demonstrated; frequent in cats
- Cluster seizures—more than one seizure in 24 hours
- Status epilepticus—continuous seizure activity, or seizures repeated at brief intervals without complete recovery between seizures; status epilepticus can be localized (known as “focal” or “non convulsive” status epilepticus) or generalized (known as “convulsive status epilepticus”)—convulsive status epilepticus is a life-threatening medical emergency

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Localized (focal) seizures with/without secondary generalization are the most frequent—movements of facial muscles predominate, such as twitches of eyelids, whiskers and ears; it may be associated with whole body trembling/shaking, leg motions, hair standing up (known as “piloerection”), dilated pupils, frantic running, and colliding with objects
- Generalized tonic-clonic motor seizures—symmetrical sustained, repetitive (known as “tonic-clonic”) contractions of leg muscles on both sides of the body and movement of the head up toward the back; often associated with salivation, urination, and defecation—by the time of admission to a veterinary hospital, the gross motor activity may have stopped, but twitching of the lids and body/limb jerks still may be present
- Injury is frequent—biting of tongue, torn nails or claws
- Mental status, reflexes, and menace response may be abnormal
- Other signs and physical examination findings vary, based on underlying cause of the seizures and the severity of the seizures

CAUSES

- Pattern of seizures (such as age of cat at onset of seizure activity, type and frequency of seizures) is the most important factor in determining possible causes

Extracranial Cause (disorder outside of the head, leading to seizure activity)

- Metabolic disorder—low blood glucose or sugar (known as “hypoglycemia”), such as from insulin overdose; low calcium levels in the blood (known as “hypocalcemia”) following surgery to remove the thyroid gland (known as “thyroidectomy”); high blood pressure (hypertension) secondary to kidney transplant; nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”)
- Poisons

Intracranial Cause (disorder inside of the head, leading to seizure activity)

- Anatomic or structural disorder—congenital (present at birth) malformation
- Metabolic disorder—storage diseases (inherited metabolic diseases in which harmful levels of materials accumulate in the body’s cells and tissues)
- Tumors or cancer—meningioma, glioma, lymphoma
- Inflammatory infectious disease—viral diseases (such as feline infectious peritonitis [FIP]), toxoplasmosis, cryptococcosis
- Trauma
- Poisons—insecticides (such as organochlorines, pyrethrins and pyrethroids); chemotherapeutic drug, chlorambucil, used in lymphoma treatment
- Blood vessel or circulatory disorders—red-blood cell (RBC) count above the normal ranges, characterized by the uncontrolled, but orderly production of excessive numbers of mature red-blood cells by the bone marrow (known as “polycythemia vera”) leading to sludging of the blood (known as “hyperviscosity”); a disorder characterized by lack of blood flow to part of the brain, caused by

migration of *Cuterebra* larva (known as “feline ischemic encephalopathy secondary to *Cuterebra* larva”)

RISK FACTORS

- Any brain lesion
- Treatment with chlorambucil, a chemotherapeutic drug
- Kidney failure
- Diabetes mellitus (“sugar diabetes”)



TREATMENT

HEALTH CARE

- Outpatient—isolated recurrent seizures in an otherwise healthy animal
- Inpatient—cluster seizures (more than one seizure in 24 hours) and status epilepticus (repeated or prolonged seizure activity)
- Constant medical supervision
- An intravenous (IV) catheter will be established to allow for drug and fluid administration
- Blood should be drawn for rapid measurement of blood gases, glucose, calcium, and levels of anti-seizure drugs (also known as “anticonvulsants”), if pet has been on anticonvulsants
- Carefully cool the body, if the cat has an elevated body temperature (known as “hyperthermia”)

SURGERY

- Surgical opening of the skull (known as a “craniotomy”)—surgical removal of tumor or cancer (meningioma or other accessible mass)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Seizure type and frequency determine therapeutic approach

Isolated Recurrent Generalized Seizures

- Medications to control seizures (known as “anti-epileptic drugs” or “anticonvulsants”)—phenobarbital, diazepam
- Initiate gradually to avoid overt sedation

Convulsive Cluster Seizures (more than one seizure in 24 hours) and Status Epilepticus (repeated or prolonged seizure activity)

- Treat cluster seizures and generalized status epilepticus early
- Medications to control seizures (anti-epileptic drugs or anticonvulsants)—phenobarbital, diazepam; choice and method of administration of medication based on status of seizure activity at time of presentation to the animal hospital

Persistent Seizures

- Propofol (an anesthetic drug) administered at doses below those needed to induce anesthesia

Other Medications

- Dexamethasone—a steroid to improve fluid build-up (known as “edema”) in the brain secondary to status epilepticus (repeated or prolonged seizure activity) and to treat the primary cause, if symptomatic epilepsy is suspected
- Gabapentin to help control seizures
- Levetiracetam to help control seizures



FOLLOW-UP CARE

PATIENT MONITORING

- Blood work (complete blood count [CBC], serum biochemistry profile) prior to initiating treatment, 4 to 6 weeks after starting phenobarbital, and repeat every 6 to 12 months
- Blood work (creatinine kinase [CK]) to evaluate muscle damage and subtle on-going seizure activity
- Measure phenobarbital serum level two weeks after initiation of treatment; dosage may be changed, based on blood test results; re-measure phenobarbital serum levels periodically until therapeutic range is reached
- Blood work (liver enzymes) 3 to 5 days after starting treatment with diazepam

POSSIBLE COMPLICATIONS

- Side effects of phenobarbital—low platelet count (known as “thrombocytopenia”), low white-blood cell count (known as “neutropenia”), itchiness (known as “pruritus”), or swollen feet; “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- Diazepam may cause sudden death of liver cells (known as “acute hepatic necrosis”)
- Cardiovascular and respiratory collapse from over dose

EXPECTED COURSE AND PROGNOSIS

- Depends on the underlying cause and response to treatment
- Cats with ‘probably symptomatic epilepsy’ have a good long-term prognosis
- Cats can recover despite episode of severe cluster seizures (more than one seizure in 24 hours) and generalized status epilepticus (repeated or prolonged seizure activity)



KEY POINTS

- Treat cluster seizures (more than one seizure in 24 hours) and generalized status epilepticus (repeated or prolonged seizure activity) early
- Anti-epileptic (anticonvulsant) treatment in symptomatic epilepsy may not help until the primary cause is addressed
- Keep a seizure calendar noting date, time, severity and length of seizures

SEIZURES (CONVULSIONS, STATUS EPILEPTICUS) IN DOGS



BASICS

OVERVIEW

- “Seizures” are periods of uncontrolled electrical activity in the brain (also known as “convulsions”); “status epilepticus” is repeated or prolonged seizure activity
- “Epilepsy”—disorder characterized by recurring seizures that originate from the brain
- “Idiopathic epilepsy”—epilepsy of unknown cause; syndrome that involves only epilepsy, with no demonstrable underlying brain lesion or other nervous system signs
- “Symptomatic epilepsy”—syndrome in which epileptic seizures are the result of identifiable, structural brain lesions
- “Probably symptomatic epilepsy”—when symptomatic epilepsy is suspected, but a lesion cannot be demonstrated
- Cluster seizures—more than one seizure in 24 hours
- Status epilepticus—continuous seizure activity, or seizures repeated at brief intervals without complete recovery between seizures; status epilepticus can be localized (known as “focal” or “non convulsive” status epilepticus) or generalized (known as “convulsive status epilepticus”)—convulsive status epilepticus is a life-threatening medical emergency
- Seizures are classified as “focal” (localized), “generalized,” and “focal with secondary generalization”

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Aura—beginning of a seizure; dog is aware or feeling changes associated with the oncoming seizure—behavioral changes may be seen (such as looking frightened, seeking owner’s assistance, or hiding); aura is not always present; it indicates localized (focal) onset of seizure activity
- Seizure—may start with an aura and progress to a convulsive generalized seizure; dog lies on its side with symmetrical sustained, repetitive (known as “tonic-clonic”) contractions of leg muscles on both sides of the body; often see salivation/drooling, urination, and/or defecation
- Period following the seizure—time of recovery that may last a few minutes to hours; signs include disorientation, confusion, aimless pacing, blindness, increased appetite (known as “polyphagia”)
- Most seizures occur when the dog is resting
- In localized (focal) seizures, the dog is conscious, but mental status may be altered
- Dog may be having seizures, may be normal or may have signs (such as disorientation, confusion) following a seizure at time of presentation to the veterinarian
- Mental status, reflexes, and menace response may be abnormal
- Other signs and physical examination findings vary, based on underlying cause of the seizures and the severity of the seizures

CAUSES

- Pattern of seizures (such as age of dog at onset of seizure activity, type and frequency of seizures) is the most important factor in determining possible causes

Extracranial Cause (disorder outside of the head, leading to seizure activity)

- Metabolic—low blood glucose or sugar (known as “hypoglycemia”); low levels of calcium in the blood (known as “hypocalcemia”); sudden (acute) kidney failure failure; nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”)
- Poisons

Intracranial Cause (disorder inside of the head, leading to seizure activity)

- Gradual deterioration, leading to loss of function (known as “degeneration”) of the brain—disorder of the brain characterized by changes of aging (known as “senile encephalopathy”)
- Metabolic disease—storage diseases (inherited metabolic diseases in which harmful levels of materials accumulate in the body’s cells and tissues)
- Tumors or cancer—primary tumors (meningioma, gliomas); secondary cancer, due to the spread of the cancer (known as “metastatic cancer”)
- Inflammatory infectious disease—viral diseases (such as canine distemper); fungal diseases; protozoal diseases (such as *Neospora*,

Toxoplasma); rickettsial diseases (such as ehrlichiosis, Rocky Mountain spotted fever)

- Of unknown cause (so called “idiopathic disease”) or immune-mediated disease—various diseases characterized by inflammation of the brain, spinal cord and their surrounding membranes (the membranes are known as “meninges”), such as granulomatous meningoencephalitis, eosinophilic meningoencephalomyelitis; pug encephalitis; necrotizing meningoencephalitis of Maltese dogs and Yorkshire terriers
- Trauma
- Blood vessel or circulatory disorders—blood clot or bleeding in the brain (known as a “cerebral vascular accident”)
- Epilepsy of unknown cause (idiopathic epilepsy)—age-related, presumed genetic
- Probably symptomatic epilepsy—following inflammation of the brain (known as “encephalitis”) or trauma (scar tissue)



TREATMENT

HEALTH CARE

- Outpatient—isolated seizures in an otherwise healthy dog
- Inpatient—cluster seizures (more than one seizure in 24 hours) and status epilepticus (repeated or prolonged seizure activity)
- Constant medical supervision
- An intravenous (IV) catheter will be established to allow for drug and fluid administration
- Blood should be drawn for rapid measurement of blood gases, glucose, calcium, and levels of anti-seizure drugs (also known as “anticonvulsants”), if pet has been on anticonvulsants
- Carefully cool the body, if the dog has an elevated body temperature (known as “hyperthermia”)

SURGERY

- Surgical opening of the skull (known as a “craniotomy”)—surgical removal of tumor or cancer (meningioma or other accessible mass)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Seizure type and frequency determine therapeutic approach

Convulsive Cluster Seizures (more than one seizure in 24 hours) and Status Epilepticus (repeated or prolonged seizure activity)

- Treat vigorously with medications to control seizures (known as “anti-epileptic drugs” or “anticonvulsants”)—diazepam, phenobarbital; choice and method of administration of medication based on status of seizure activity at time of presentation to the animal hospital

Persistent Seizures

- Propofol (an anesthetic drug), generally administered at doses below those needed to induce anesthesia

Other Medications

- Dexamethasone—a steroid to improve fluid build-up in the brain (known as “cerebral edema”) secondary to status epilepticus (repeated or prolonged seizure activity)
- Steroids—for treatment of fluid build up in the brain (cerebral edema) secondary to severe inflammatory central nervous system disease, even if caused by an infectious agent
- Potassium bromide—used to control seizures; requires a prolonged period to reach therapeutic levels; therefore, it is not indicated in the treatment of convulsive status epilepticus (repeated or prolonged seizure activity)
- Pentobarbital (an anesthetic drug)—for patients that fail to respond to diazepam and phenobarbital; antiepileptic activity of propofol is superior to that of pentobarbital

Localized (Focal) Status Epilepticus (repeated or prolonged seizure activity)

- Medications to control seizures (anti-epileptic drugs or anticonvulsants)—diazepam, phenobarbital; effective for localized (focal) and generalized seizures
- Potassium bromide—in people, more effective against generalized seizures
- Newer medications to control seizures in people are preferable to older medications, since most are developed to control localized (focal) seizures



FOLLOW-UP CARE

PATIENT MONITORING

- Inpatients—constant supervision for monitoring of seizure activity

POSSIBLE COMPLICATIONS

- Phenobarbital—liver toxicity after long-term treatment; sudden (acute) low white-blood cell count (known as “neutropenia”)—rare side effect, seen in the first few weeks of use; if it occurs, permanently discontinue treatment with phenobarbital (as directed by your pet’s veterinarian)
- Continued seizures, despite adequate serum levels of medications to control seizures (anti-epileptic drugs or anticonvulsants)
- Permanent nervous system deficits (such as blindness or abnormal behavior) may follow severe status epilepticus (repeated or prolonged seizure activity)
- Generalized status epilepticus (repeated or prolonged seizure activity) may lead to elevated body temperature (known as “hyperthermia”), acid–base and electrolyte imbalances, fluid build-up in the lungs (known as “pulmonary edema”), circulatory collapse and death

EXPECTED COURSE AND PROGNOSIS

- Epilepsy of unknown cause (idiopathic epilepsy) represents a large proportion of dogs with generalized status epilepticus (repeated or prolonged seizure activity)
- Dogs with inflammation of the brain and its surrounding membranes (known as “meningoencephalitis”) that develop generalized status epilepticus have the poorest outcome
- Eyelid or lip twitching in a heavily sedated patient is a sign of ongoing seizure activity
- Pet may need 7 to 10 days before returning to normal after status epilepticus (repeated or prolonged seizure activity); vision returns last



KEY POINTS

- Treat cluster seizures (more than one seizure in 24 hours) and generalized status epilepticus (repeated or prolonged seizure activity) early
- Anti-epileptic (anticonvulsant) treatment in symptomatic epilepsy may not help until the primary cause is addressed
- Keep a seizure calendar noting date, time, severity and length of seizures
- Ask your pet’s veterinarian for an in-home emergency plan for cluster seizures

SEPARATION-ANXIETY SYNDROME



BASICS

OVERVIEW

- A distress response that dogs may experience when separated from the person or persons to whom they are most attached
- This distress may result in problem behaviors in the absence or perceived absence of the person(s), including episodes of destruction, vocalization, and elimination
- Separation anxiety is a subset of separation-related problems that may have different underlying motivations, including fear, anxiety, over-attachment to the person(s), and lack of appropriate stimulation or interactions

GENETICS

- None known

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Primarily dogs; speculated that 7% to 28% of companion dogs experience some degree of separation-anxiety syndrome
- Possible in cats

Mean Age and Range

- Any age, most commonly in dogs greater than 6 months of age; number of cases may increase in dogs greater than 8 years of age

Predominant Sex

- None recorded

SIGNS/OBSERVED CHANGES in the ANIMAL

- Destruction, vocalization (whining, howling, barking) and indoor elimination are reported most commonly; destruction often targets windows and doors and/or the person's possessions
- Other signs include behavioral depression, lack of appetite (anorexia), drooling, hiding, shaking, panting, pacing, attempts to prevent the person's departure, and self-trauma from lick lesions; occasionally diarrhea, while the person is gone
- Signs of strong pet-person attachment, usually excessive attention-seeking behaviors and following behaviors; excited and prolonged greeting behavior upon the person's return, regardless of the length of the absence
- In cats, elimination problems in the person's absence may be linked to separation-related anxiety
- Separation distress behavior(s) occurs regardless of the length of the person's absence and tend to occur within 30 minutes of the person's departure, but may occur on and off all day
- Specific triggers may be identified, such as the person getting keys, putting on outer garments, or packing the car
- May occur on every departure and absence or only with atypical departures (such as after-work, evening, or weekend departures); the reverse pattern also may be seen
- Physical examination usually is normal
- Injuries possible in escape attempts or while engaging in destructive activities
- Skin lesions from excessive licking
- Rare cases of dehydration from drooling or diarrhea due to stress

CAUSES

- Specific causes are unknown
- Speculated causes include the following:
 - Improper socialization to the person's departure and absence
 - Lack of appropriate pet-person interactions
 - Prolonged contact with humans, without learning to be alone
 - Traumatic episodes during the person's absence
 - Decline in thinking, learning, and memory, frequently associated with aging (known as "cognitive decline")

RISK FACTORS

- Suspected, but not proven, risk factors include adoption from humane shelters, periods of extended time with preferred person (such as during vacation or illness), boarding, lack of detachment when young

- Senior animals
- Possible correlation between separation anxiety and noise phobias (such as thunderstorm phobias)



TREATMENT

HEALTH CARE

Changing the Pet's Perception of Pre-Departure Cues

- Repeat pre-departure cues (such as picking up keys, walking to the door) without leaving
- Repeat 2 to 4 times daily, until the dog does not respond to cues with anxious behaviors (such as panting, pacing, following, or increased vigilance)
- Goal is to remove the dog's association with the cues and the person's departures, and to diminish the anxious response

Counterconditioning (Response Substitution)

- Teach the dog to "sit/stay" near the typical exit door
- Gradually increase the distance between the dog and the person toward the door
- The person slowly progresses toward the door, increasing the time away on each trial
- Eventually elements of departure, such as opening and closing the door, are added
- Finally, the person steps outside the door and returns

Classical Counterconditioning

- Leave the dog a delectable food treat or food-stuffed toy on departure
- Associate departure with something pleasant

Changing Departure and Return Routine

- Ignore the pet for 15 to 30 minutes prior to departure and upon return
- On return, attend to the dog only when it is calm and quiet; however, allow the dog outside to eliminate

Independence Training

- Teach the dog to be more independent of the person(s)
- All attention is at person's initiation—person begins and ends attention sessions, rather than the dog initiating attention
- No attention on pet demand
- Attention must be earned by the pet by performing a task, such as "sit"
- Decrease following behavior while the person is at home
- Teach the dog to stay in another location, away from the person

Graduated Planned Departures and Absences

- Begun after dog does not respond to pre-departure cues with anxious behaviors
- Use short absences to teach the dog how to be left home alone
- Departures must be short enough not to elicit a separation distress response; initial departures must be very short, 1 to 5 minutes
- Goal—animal learns consistency of person's return and to experience departure and absence without anxiety
- Departures must be just like real departures (person must do all components of departure, including leaving in the car, if that is how they usually depart); person will leave a safety cue (such as leaving a radio or television on, ringing a bell) on planned departures only (must not be used on departures where length of absence is not controlled, such as work departures)
- Length of absence is slowly increased at 3 to 5 minute intervals, if no signs of distress were evident at the shorter interval; increase in interval must be variable; intersperse short (1 to 3 minute) with longer (5 to 20 minute) departures
- If destruction, elimination, or vocalization occurs, departure was too long
- If departures and absences are continued, even though distress behaviors are present, the dog will get worse
- Once the pet can be left for 2 to 3 hours on a planned departure, it often can be left all day
- Safety cue is slowly phased out over time, or can be used indefinitely

Arrangements for the Pet During Retraining and Person's Absence

- Allow no more destructive activity, if possible
- Mixing up or eliminating triggering departure cues may help diminish the anxious responses
- Doggy daycare arrangements or pet sitters
- Gradual conditioning to a crate; crates are not recommended, unless the dog is already crate-trained and comfortable being left in a crate

ACTIVITY

- Regular, scheduled daily exercise and playtime are beneficial

DIET

- No dietary changes are necessary, unless animal also has diarrhea



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Clomipramine—a tricyclic antidepressant (TCA)—drug of first choice and only drug approved for use in the treatment of separation anxiety in dogs; approved for dogs older than 6 months of age; must be given daily, not on an “as needed” basis, as it may take 2 to 4 weeks before behavioral effect is evident
- Tricyclic antidepressants (TCAs), such as amitriptyline
- Selective serotonin reuptake inhibitors (SSRIs), such as fluoxetine
- Benzodiazepines, such as alprazolam
- D.A.P.® (Dog-Appeasing Pheromone); a synthetic analogue of the natural appeasing pheromones of the nursing bitch, which calm puppies; used to calm dogs in fearful, stressful, and anxiety situations (such as separation anxiety and noise phobias); available as a plug-in diffuser and collar



FOLLOW-UP CARE

PATIENT MONITORING

- Good client follow-up is necessary to monitor both the behavioral treatment plan and medication, if prescribed
- Weekly follow-up is best in the early stages to assess effectiveness of the treatment plan and compliance with instructions—once the dog has become more independent, has become less responsive to pre-departure cues, and is calmer on departures and returns, graduated planned departures may be implemented

PREVENTIONS AND AVOIDANCE

- Teaching animals how to be left home alone, making animals independent

POSSIBLE COMPLICATIONS

- Injuries during escape attempts and ongoing destruction and elimination disrupt the human-animal bond and may result in relinquishment of the pet to an animal shelter or animal control facility

EXPECTED COURSE AND PROGNOSIS

- Separation anxiety often responds well to behavioral modification, with or without medication
- Some severe cases can be very resistant to treatment; other behavioral disorders occurring at the same time may make resolution more difficult
- Drug therapy alone is rarely curative for most behavioral disorders; realistically, drug therapy can be expected to decrease the anxiety associated with the person's departure, but the dog still must be taught how to be left alone during the person's absences



KEY POINTS

- Have realistic expectations of the time course of treatment and the need for behavior modification, in order to have successful resolution of the problem
- Drug therapy alone is rarely curative for most behavioral disorders; realistically, drug therapy can be expected to decrease the anxiety associated with the person's departure, but the dog still must be taught how to be left alone during the person's absences
- Problem behavior may take weeks or months to resolve, depending on severity and duration of the problem

GENERALIZED BACTERIAL INFECTION (SEPSIS) AND THE PRESENCE OF BACTERIA IN THE BLOOD (BACTEREMIA)



BASICS

OVERVIEW

- “Sepsis”—generalized (systemic) response to bacterial infection (such as fever or low blood pressure [known as “hypotension”])
- “Bacteremia”—the presence of bacteria in the blood

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Predominant Sex

- Large-breed male dogs—more susceptible to developing bacterial infection/inflammation of the lining of the heart (known as “bacterial endocarditis”) and bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition known as “diskospondylitis”)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs may be sudden (acute) or may occur in a vague or episodic fashion
- Variable and may involve multiple organ systems
- More severe when gram-negative bacteria are involved
- Dogs that develop an overt generalized (systemic) response to bacterial infection (sepsis)—earliest signs usually involve the gastrointestinal tract (including the stomach and intestines)
- Cats—usually involves the respiratory system (including the lungs)
- Intermittent or persistent fever
- Lameness
- Depression
- Rapid heart rate (known as “tachycardia”)
- Heart murmur
- Low blood pressure (hypotension)
- Weakness

CAUSES

- Dogs—bacteria; gram-negative bacteria (especially *E. coli*) most common; gram-positive cocci and obligate anaerobes also are important; infection with more than one type of bacteria (known as “polymicrobial infection”) reported in about 20% of dogs with positive blood cultures (that is, bacteria are grown in the laboratory from samples of blood); “obligate anaerobes” are bacteria that must live and grow in the absence of oxygen
- Cats—bacteria; disease-causing agents usually are gram-negative bacteria or obligate anaerobes; *Salmonella* is the most common gram-negative bacteria cultured
- *Pseudomonas aeruginosa*—uncommon isolate from animal blood cultures

RISK FACTORS

- Very sudden (known as “peracute”) disease—most often associated with infection/inflammation of the uterus with accumulation of pus (known as “pyometra”) and disruption of the gastrointestinal tract
- More prolonged onset of disease—associated with infections of the skin, upper urinary tract (kidneys and ureters), mouth, or prostate
- Excessive levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”); diabetes mellitus (“sugar diabetes”); liver or kidney failure; surgical removal of the spleen (known as “splenectomy”); cancer; and burns
- Inability to develop a normal immune response (known as “immunodeficiency”)—chemotherapy; feline immunodeficiency virus (FIV); surgical removal of the spleen (splenectomy)
- Administration of steroids—considered an important risk factor for the presence of bacteria in the blood (bacteremia); allows greater multiplication of bacteria in body tissues
- Intravenous catheter—provides rapid access for bacteria to enter the bloodstream
- Indwelling urinary catheters



TREATMENT

HEALTH CARE

- Successful treatment requires early identification of the problem and aggressive intervention; careful monitoring is essential—patient status may change rapidly
- Low blood pressure (hypotension)—intravenous fluids (such as lactated Ringer’s solution)
- Colloids (fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume and expand blood volume), such as hydroxyethyl starch (hetastarch)
- Low blood glucose or sugar (hypoglycemia)—may add dextrose to intravenous fluids
- Electrolytes and acid–base balance—correct abnormalities
- Abscesses—locate and drain
- External sources of infection—give appropriate attention to wound care and bandage changes
- Internal sources of infection (such as infection/inflammation with accumulation of pus in the uterus [pyometra] or disruption of the bowel)—surgical intervention is essential

DIET

- Nutritional support—provide by assisted feeding or placement of a feeding tube

SURGERY

- Surgery may be required for treatment of certain disorders (such as infection/inflammation with accumulation of pus in the uterus [pyometra] or disruption of the bowel) and to establish drainage for abscesses



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics—usually selected before bacterial culture and sensitivity test results are available; do not delay treatment while waiting for test results; direct therapy to cover all possible types of bacteria (gram-positive and negative bacteria; aerobic and anaerobic bacteria); “aerobic bacteria” are bacteria that can live and grow in the presence of oxygen; “anaerobic bacteria” are bacteria that can live and grow in the absence of oxygen
- Antibiotics—administered intravenously
- If patient is not in shock—a good choice of antibiotics is a first-generation cephalosporin, such as cefazolin
- Aminoglycosides—(class of antibiotics) may be added to the treatment protocol, if more aggressive therapy is warranted; example is gentamicin



FOLLOW-UP CARE

PATIENT MONITORING

- Aminoglycoside therapy—monitor kidney function
- Blood pressure and electrocardiogram to monitor circulation and the heart

POSSIBLE COMPLICATIONS

- Gram-negative generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”)—high death rate; death owing to low blood pressure (hypotension), electrolyte and acid–base disturbances, and shock due to the presence of bacterial toxins in the blood (known as “endotoxic shock”)

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause
- Generalized (systemic) response to bacterial infection (sepsis) and the presence of bacteria in the blood (bacteremia) are serious conditions, which may be fatal



KEY POINTS

- Generalized (systemic) response to bacterial infection (sepsis) and the presence of bacteria in the blood (bacteremia) are serious conditions, which may be fatal
- Successful treatment requires early identification of the problem and aggressive intervention; careful monitoring is essential—patient status may change rapidly
- Do not delay treatment while waiting for bacterial culture and sensitivity test results
- Direct therapy to cover all possible types of bacteria (gram-positive and negative bacteria; aerobic and anaerobic bacteria); “aerobic bacteria” are bacteria that can live and grow in the presence of oxygen; “anaerobic bacteria” are bacteria that can live and grow in the absence of oxygen

SEPTIC SHOCK



BASICS

OVERVIEW

- Shock associated with generalized bacterial infection (generalized bacterial infection known as “sepsis;” condition known as “septic shock”)
- Develops as a complication of overwhelming generalized (systemic) infection
- Septic shock is associated with low blood flow (known as “hypoperfusion”) or low blood pressure (known as “hypotension”) that may or may not respond to fluids or medical treatment to maintain arterial blood pressure

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Possible history of known infection (such as urinary tract infection [UTI] or infection/inflammation of the prostate [known as “prostatitis”])
- Previous surgery possible
- Signs from other conditions or treatments that potentially decrease the immune response (known as “immunosuppressive” conditions or treatments), such as diabetes mellitus (“sugar diabetes”); increased levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”), or chemotherapy regimens

Early or Compensatory Shock

- Rapid heart rate (known as “tachycardia”)
- Normal or high arterial blood pressure
- Bounding pulses
- Reddened moist tissues of the body (known as “hyperemic mucous membranes”)
- The pink or red color of the gums is very quick to return when the gums are blanched by finger pressure (known as “rapid capillary refill time”)
- Fever
- Rapid breathing (known as “tachypnea”)

Late or Decompensatory Shock

- Rapid heart rate (tachycardia) or slow heart rate (known as “bradycardia”)
- Poor pulses
- Pale gums or moist tissues of the body (known as “mucous membranes”)
- The pink color of the gums is slow to return when the gums are blanched by finger pressure (known as “poor capillary refill time”)
- Cool extremities
- Low body temperature (known as “hypothermia”)
- Mental depression or stupor
- Production of only small amounts of urine (known as “oliguria”)
- Difficulty breathing (known as “dyspnea”); rapid breathing (tachypnea)
- Small, pinpoint areas of bleeding (known as “petechia”) in the skin and moist tissues of the body (mucous membranes)
- Fluid build-up in the tissues, especially the legs and under the skin (known as “peripheral edema”)
- Gastrointestinal bleeding
- Extreme weakness

CAUSES

- Compromise of the lining of the gastrointestinal tract, resulting in bacteria moving from the intestinal tract into the body and causing bacterial toxins to accumulate in the blood (known as “endotoxemia”)
- Urinary tract infection
- Infection/inflammation of the prostate (prostatitis) and abscesses of the prostate
- Gastrointestinal rupture

- Bacterial infection of the lining of the abdomen (known as “septic peritonitis”)
- Pneumonia
- Bacterial infection of the lining of the heart (known as “bacterial endocarditis”)
- Bite wounds

RISK FACTORS

- Coexistent condition or treatment causing decrease in the immune response and increasing likelihood of development of generalized bacterial infection (sepsis); conditions include such diseases as diabetes mellitus (“sugar diabetes”); increased levels of steroids produced by the adrenal glands (hyperadrenocorticism or Cushing’s disease); and treatment with high-dosage steroids or chemotherapy
- Old or young age



TREATMENT

HEALTH CARE

- Inpatient because of circulatory collapse
- Vigorous fluid therapy is needed to increase effective circulating blood volume; crystalloids are fluids that contain electrolytes (chemical compounds, such as sodium, potassium, chloride) necessary for the body to function, crystalloids generally are similar to the fluid content (plasma) of the blood and move easily between the blood and body tissues, example is lactated Ringer’s solution; colloids are fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch
- Oxygen supplementation—as important as fluid replacement; administer by oxygen cage, mask, or nasal cannula

SURGERY

- Surgically remove any source of generalized bacterial infection (sepsis), such as an abscess; aggressive treatment and life support may be required



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Septic shock that does not respond to medical treatment (known as “refractory septic shock”)—systemic blood pressure may be raised through the use of medications that improve heart muscle contraction (known as “positive inotropes,” such as dobutamine) or medications to constrict blood vessels to increase blood pressure (known as “vasopressors,” such as dopamine, vasopressin, phenylephrine or norepinephrine)
- Broad-spectrum antibiotics administered intravenously are essential; while awaiting results of blood, urine, or tissue bacterial cultures, treatment may include one of the following antibiotic combinations: ampicillin or cephalexin and gentamicin or enrofloxacin; metronidazole can be used with either of these combinations
- Sodium bicarbonate may be given intravenously to a patient with severe metabolic acidosis (a condition in which levels of acid are increased in the blood) to improve blood pH level



FOLLOW-UP CARE

PATIENT MONITORING

- Heart rate, pulse intensity, color of gums and moist tissues (mucous membrane), breathing rate, lung sounds, urine output, mental status; and rectal temperature during aggressive treatment with fluids or medications that improve heart muscle contraction (positive inotropes)
- Electrocardiogram (“ECG,” a recording of the electrical activity of the heart) and blood pressure measurement are useful; use blood-gas analysis (measurements of oxygen and carbon dioxide levels in arterial blood) and pulse oximetry (a means of measuring oxygen levels in blood), to monitor tissue oxygen levels

- Blood work (such as packed cell volume [“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood]; serum total protein [a quick laboratory test that provides general information on the level of protein in the fluid portion of the blood]; serum electrolytes; liver enzymes; blood urea nitrogen; and serum creatinine)

POSSIBLE COMPLICATIONS

- Electrolyte and acid–base disturbances
- Irregular heart beats (known as “arrhythmias”)
- Fluid build-up in the lungs (known as “pulmonary edema”) or acute respiratory distress syndrome (“ARDS,” a group of lung abnormalities that develop secondary to various serious illnesses that cause sudden breathing difficulties)
- Blood clots to the lungs (known as “pulmonary thromboembolism”)
- Blood-clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”)
- Kidney dysfunction
- Liver dysfunction
- Reduced blood flow to part of the gastrointestinal tract, usually due to some type of blockage in a blood vessel, leading to decreased oxygen in the tissues (condition known as “gastrointestinal ischemia”) and movement of bacteria from the gastrointestinal tract into the body
- Fluid build-up in the brain (known as “cerebral edema”) and seizures
- Inflammation of the pancreas (known as “pancreatitis”)
- Inflammation of blood vessels (known as “vasculitis”)
- Fluid build-up in the tissues, especially the legs and under the skin (peripheral edema)
- Cardiac arrest
- Death

EXPECTED COURSE AND PROGNOSIS

- Depend on underlying cause
- Septic shock is a life-threatening condition



KEY POINTS

- Septic shock is a life-threatening condition
- Septic shock is associated with low blood flow (hypoperfusion) or low blood pressure (hypotension) that may or may not respond to fluids or medical treatment to maintain arterial blood pressure
- Vigorous fluid therapy is needed to increase effective circulating blood volume
- Oxygen supplementation—as important as fluid replacement
- Broad-spectrum antibiotics administered intravenously are essential
- Aggressive treatment and life support may be required

SHOULDER JOINT—LIGAMENT AND TENDON CONDITIONS



BASICS

OVERVIEW

- The shoulder joint is a “ball-and-socket” joint, made up of bones (the scapula or shoulder blade and the humerus or upper bone of the front leg) that is supported by ligaments and tendons
- A “ligament” is a band of connective or fibrous tissue that connects two bones or cartilage at a joint; a “tendon” is a band of connective or fibrous tissue that connects a muscle to a bone
- Shoulder-joint ligament and tendon conditions make up the majority of causes for lameness in the canine shoulder joint, excluding osteochondritis dissecans (condition characterized by abnormal development of bone and cartilage, leading to a flap of cartilage within the joint)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Medium- to large-breed dogs

Mean Age and Range

- Skeletally mature dogs 1 year of age or older
- Usually 3 to 7 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on severity and long-term nature (chronicity) of the disease
- Decrease in muscle mass (known as “muscle atrophy”)—consistent finding for all conditions
- Bicipital tenosynovitis (inflammation of the tendon and surrounding sheath of the biceps tendon)—onset usually subtle; often of several months’ duration; trauma may be the inciting cause; subtle, intermittent lameness that worsens with exercise; short and limited swing phase of gait owing to pain on extension and flexion of the shoulder; pain inconsistently demonstrated on manipulation of shoulder
- Rupture of the tendon of the biceps brachii muscle—signs similar to bicipital tenosynovitis; may have sudden (acute) onset due to a known traumatic event; usually subtle, long-term (chronic) lameness that worsens with exercise
- Mineralization of the tendon of the supraspinatus muscle—onset usually subtle; long-term (chronic) lameness that worsens with activity
- Forcible separation (known as an “avulsion”) or fracture of the tendon of the supraspinatus muscle—signs similar to mineralization of supraspinatus tendon
- Deterioration and scarring (known as “fibrotic contracture”) of the infraspinatus muscle—usually sudden (acute) onset during a period of outdoor exercise (such as hunting); shoulder lameness and tenderness gradually disappear within 2 weeks; condition results in long-term (chronic), persistent lameness 3 to 4 weeks later, which is not particularly painful; decrease in muscle mass of the infraspinatus muscle (muscle atrophy); when patient is walking—lower limb swings in an arc away from the body, as the paw is advanced

CAUSES

- Indirect or direct trauma—likely
- Repetitive strain injury (indirect trauma)—most common

RISK FACTORS

- Overexertion and/or fatigue
- Poor conditioning before performing athletic activities
- Obesity



TREATMENT

HEALTH CARE

- Outpatient—early diagnosis
- Inpatient—long-term (chronic), severe disease requires surgical intervention
- Bicipital tenosynovitis (inflammation of the tendon and surrounding sheath of the biceps tendon)—50% to 75% success with medical treatment; requires surgery with evidence of long-term (chronic) changes and failure of response to medical management
- Rupture of the tendon of the biceps brachii muscle generally requires surgery
- Mineralization of the tendon of the supraspinatus muscle—may be an incidental finding; requires surgery after excluding other causes of lameness and attempting medical treatment
- Forcible separation (avulsion) or fracture of the tendon of the supraspinatus muscle—often requires surgery because of persistent bone-fragment irritation of the tendon
- Deterioration and scarring (fibrotic contracture) of the infraspinatus muscle—requires surgery
- Ice packing (known as “cryotherapy”)—immediately following surgery; helps reduce inflammation and swelling at the surgery site; performed 5 to 10 minutes every 8 hours for 3 to 5 days or as directed by your pet’s veterinarian
- Regional massage and range-of-motion exercises—improve flexibility; decrease loss of muscle mass (muscle atrophy)

ACTIVITY

- Medical treatment—requires strict confinement for 4 to 6 weeks; premature return to normal activity likely worsens signs and leads to a long-term (chronic) condition
- Following surgery—depends on procedure performed; your pet’s veterinarian will provide instructions regarding postoperative activity and restrictions

DIET

- Weight control—decreases stress placed on arthritic joints

SURGERY

- Bicipital tenosynovitis (inflammation of the tendon and surrounding sheath of the biceps tendon)—recommended with poor response to medical treatment and long-term (chronic) disease; goal is to eliminate movement of the biceps tendon within the inflamed synovial sheath
- Rupture of the tendon of the biceps brachii muscle—reattach tendon to the humerus (upper bone in the front leg) with a screw and spiked washer or pass the tendon through a bone tunnel and suture it to the tendon of the supraspinatus muscle
- Mineralization of the tendon of the supraspinatus muscle—surgically cut into the tendon; remove the calcium (mineral) deposits
- Forcible separation (avulsion) or fracture of the tendon of the supraspinatus muscle—remove the bone fragment(s)
- Deterioration and scarring (fibrotic contracture) of the infraspinatus muscle—surgical division of the tendon and removal of part of the tendon; the surgeon often will feel a distinct “pop” after removal of the last scar tissue; procedure should allow complete range of motion of the shoulder joint



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Nonsteroidal anti-inflammatory drugs (NSAIDs) to decrease pain and inflammation—examples are carprofen, deracoxib, etodolac, meloxicam, tepoxalin
- Medications intended to slow the progression of arthritic changes and protect joint cartilage (known as “chondroprotective drugs”), such as polysulfated glycosaminoglycans, glucosamine, and chondroitin sulfate—may help limit cartilage damage and degeneration; may help alleviate pain and inflammation

Bicipital Tenosynovitis (inflammation of the tendon and surrounding sheath of the biceps tendon)

- Injection of steroids (such as prednisolone acetate) into the joint (known as “intra-articular injection”)—initial treatment of choice
- Nonsteroidal anti-inflammatory drugs or steroids administered by mouth (known as “systemic treatment”)—not as effective
- Lameness markedly improved, but not eliminated—give a second intra-articular injection in 3 to 6 weeks
- Incomplete resolution—recommend surgery



FOLLOW-UP CARE

EXPECTED COURSE AND PROGNOSIS

- Most patients require a minimum of 1 to 2 months of rehabilitation after treatment
- Medically managed bicipital tenosynovitis (inflammation of the tendon and surrounding sheath of the biceps tendon)—often successful after one or two treatments (50% to 75% of cases) with no long-term (chronic) changes
- Surgically treated bicipital tenosynovitis (inflammation of the tendon and surrounding sheath of the biceps tendon)—good to excellent results (90% of cases); recovery to full function may take 2 to 8 months
- Surgically treated rupture of the tendon of the biceps brachii muscle—good to excellent prognosis; more than 85% of patients show improved return to function.
- Surgically treated mineralization of the tendon of the supraspinatus muscle—good to excellent prognosis; recurrence possible, but uncommon
- Surgically treated forcible separation (avulsion) or fracture of the tendon of the supraspinatus muscle—good to excellent prognosis; recurrence possible, but uncommon
- Surgically treated deterioration and scarring (fibrotic contracture) of the infraspinatus muscle—good to excellent prognosis; patients uniformly return to normal limb function

SICK SINUS SYNDROME

(TYPE OF IRREGULAR HEART BEAT)



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinus or sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- The normal heart rate for dogs varies based on the size of the dog; however, the general range is 60 to 180 beats per minute (with smaller dogs have faster normal heart rates)
- “Sick sinus syndrome” is a disorder of electrical impulse formation within, and conduction out of, the sinus node; it also affects the specialized electrical conduction system of the top two chambers of the heart (atria), the atrioventricular (AV) node, and the bottom two chambers of the heart (ventricles)
- An electrocardiogram (“ECG”) is a recording of the electrical impulse activity of the heart; the normal ECG is a tracing with P, QRS, and T waves; the P waves are the first upward deflection of the ECG tracing that look like a “bump” in the tracing; the P waves are a measure of the electrical activity of the atria; the QRS looks like an exaggerated “W” with the Q wave being a short, downward deflection, the R being a tall, spiked upward deflection, and the S being another short, downward deflection; the QRS is a measure of the electrical activity of the ventricles; finally the T wave may be an upward or downward deflection of the ECG tracing; the T wave is a measure of ventricular recovery prior to the next contraction
- Irregular heart beats (known as “arrhythmias”) noted with sick sinus syndrome include any or all of the following: inappropriate slow heart rate (known as “sinus bradycardia”); disorder in which the sinoatrial node does not generate an electrical impulse or the impulse does not leave the sinus node (known as “sinus arrest”), which causes a pause in the heart beats; condition in which the electrical impulse from the sinoatrial node is blocked from causing the atria to contract (known as a “sinoatrial exit block”); slow atrial rhythm when another part of the atrium acts as the “pacemaker” (known as a “slow ectopic atrial rhythm”); or alternating periods of slow heart rate (sinus bradycardia) and rapid heart rate (caused by electrical impulses that originate from a site other than the sinoatrial node, such as the muscle of the atria or the atrioventricular [AV] node; condition known as “supraventricular tachycardia”)
- Prolonged periods of lack of sinus node activity and often lack of atrioventricular node activity as well, leading to slow heart rate (bradycardia) may alternate with sudden onset of very fast heart rate originating in the ventricles, causing the heart to beat ineffectively (known as “paroxysmal ventricular tachycardia”), producing “bradycardia–tachycardia syndrome,” a variant of sick sinus syndrome
- P waves and QRS complexes are usually normal
- P waves may be abnormal or absent in some cases

GENETICS

- May be inherited in miniature schnauzers and West Highland white terriers

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilections

- Miniature schnauzers
- Noted commonly in cocker spaniels, dachshunds, and West Highland white terriers

Mean Age and Range

- Most dogs are greater than 6 years of age

Predominant Sex

- Female

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs vary from no clinical signs (that is, being “asymptomatic”) to weakness, fainting (known as “syncope”), collapse, and/or seizures

- Sudden death is infrequent
- Heart rate may be abnormally slow (bradycardia) or abnormally rapid (tachycardia)
- Pauses between heart beats may be noted
- Some patients appear normal

CAUSES

- Unknown cause (so called “idiopathic disease”)
- Familial (runs in certain families or lines of animals) in miniature schnauzers
- Spread of cancer (known as “metastatic disease”)
- Disease characterized by reduced blood flow to the heart, usually due to some type of blockage in a blood vessel, leading to decreased oxygen in the tissues (known as “ischemic disease”)



TREATMENT

HEALTH CARE

- Hospitalization rarely necessary, except for electrophysiologic testing of the electrical conduction system of the heart or pacemaker implantation
- Treatment is not necessary for pets that have no clinical signs (asymptomatic pets)

ACTIVITY

- Avoid vigorous exercise and stressful situations

DIET

- Modifications unnecessary

SURGERY

- Permanent artificial pacemaker necessary for dogs failing to respond to medical treatment and those exhibiting unacceptable medication side effects
- Permanent artificial pacemaker usually required for dogs with slow heart rate-rapid heart rate syndrome (bradycardia-tachycardia syndrome)
- Presence of significant heart-valve disease has implications for type of permanent pacing mode selected



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Symptomatic dogs are grouped into those showing primarily slow heart rate (bradycardia), those in which the sinoatrial node does not generate an electrical impulse or the impulse does not leave the sinus node (sinus arrest), those in which the electrical impulse from the sinoatrial node is blocked from causing the atria to contract (sinoatrial exit block) and those with a rapid heart rate (caused by electrical impulses that originate from a site other than the sinoatrial node, such as the muscle of the atria or the atrioventricular [AV] node (supraventricular tachycardia) followed by sinus arrest
- Atropine-responsive symptomatic dogs with slow heart rates (bradycardia) or in which the sinoatrial node does not generate an electrical impulse or the impulse does not leave the sinus node (sinus arrest)—medications to increase the heart rate (known as “anticholinergic drugs”), such as propantheline or hyoscyamine
- Dogs with slow heart rates (bradycardia) and in which the sinoatrial node does not generate an electrical impulse or the impulse does not leave the sinus node (sinus arrest)—may try theophylline (Theo-Dur®), terbutaline, or hydralazine, if medications to increase the heart rate (anticholinergic drugs) are ineffective
- Dogs with slow heart rate-rapid heart rate (bradycardia-tachycardia) that have clinical signs due to rapid heart rate (tachycardia) or rapid heart rate (tachycardia)-induced sinus arrest, in which the sinoatrial node does not generate an electrical impulse or the impulse does not leave the sinus node—can give heart medications (digoxin or atenolol) in attempt to suppress the rapid heart rate (supraventricular tachycardia); monitor closely for worsening of slow heart rate (bradycardia)



FOLLOW-UP CARE

PATIENT MONITORING

- Electrocardiogram (“ECG,” a recording of the electrical activity of the heart) in pets that have no clinical signs (asymptomatic pets) —to detect progression of disease
- Electrocardiogram (ECG) in pets treated medically or with a pacemaker implantation

POSSIBLE COMPLICATIONS

- Rarely, reduced blood flow to the brain or kidneys results in long-term (chronic) brain damage or kidney dysfunction, respectively

EXPECTED COURSE AND PROGNOSIS

- Good prognosis following pacemaker implantation in pets without congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Medical management—often ineffective; initial beneficial effects often not sustained



KEY POINTS

- Medical management often is ineffective
- Permanent artificial pacemaker frequently is necessary to treat a dog with sick sinus syndrome
- Prognosis is good for affected dogs following pacemaker implantation, if the dog does not have signs of congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

SMALL INTESTINAL BACTERIAL OVERGROWTH (SIBO)



BASICS

OVERVIEW

- A clinical syndrome caused by an increase in number of bacteria in the small intestine or a shift in types of bacteria in the small intestine
- Currently, no consensus has been reached on the definition and diagnostic criteria for small intestinal bacterial overgrowth (SIBO)
- Small intestinal bacterial overgrowth (SIBO) differs from infection of the intestinal tract by known disease-causing bacteria (such as *Salmonella*, *Campylobacter jejuni*, toxigenic *Clostridium perfringens*, or others)
- Two other conditions, “antibiotic-responsive diarrhea” and “tylosin-responsive diarrhea,” may refer to the same syndrome as small intestinal bacterial overgrowth (SIBO); however, all three conditions are defined using different sets of diagnostic criteria

GENETICS

- No genetic basis for small intestinal bacterial overgrowth (SIBO) has been established
- Some breeds (such as the German Shepherd dog and Chinese shar pei) appear to be at an increased risk of developing SIBO, compared to other breeds
- A genetic susceptibility for an abnormality in cell-mediated immune response to normal intestinal bacteria is suspected in people and also may occur in dogs; however, this has not been proven

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Cats may develop small intestinal bacterial overgrowth

Breed Predilections

- Subjectively, German Shepherd dogs and Chinese shar peis appear to have an increased incidence of small intestinal bacterial overgrowth (SIBO) compared to other breeds

Mean Age and Range

- Unknown
- Can be diagnosed in dogs of any age (age range, less than 1 year of age to greater than 8 years of age)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs of small intestinal disease
- Long-term (chronic) loose stools or diarrhea—common
- Weight loss, despite a reasonable appetite—common
- Rumbling or gurgling sounds caused by movement of gas in the intestinal tract (known as “borborygmus”) and excessive gas formation in the stomach or intestines (known as “flatulence”)—common
- Vomiting—occasional/variable
- Poor body condition
- Clinical signs of the underlying disease process may be seen in secondary small intestinal bacterial overgrowth (SIBO)
- Intestinal thickening is not typical unless small intestinal bacterial overgrowth (SIBO) is secondary to a disease characterized by infiltration of abnormal cells into the intestinal tissue
- Clinical signs may vary in intensity (they may increase and decrease over time [known as a “waxing and waning” course]) or be continuous
- Generalized (systemic) signs of infection (such as fever and depression) usually do not occur

CAUSES

- Unknown cause (so called “idiopathic disease”) or primary small intestinal bacterial overgrowth (SIBO)
- “Secondary small intestinal bacterial overgrowth” (SIBO) in which the increase in number of bacteria occurs due to an underlying intestinal problem—more common
- Abnormal small intestinal anatomy—may be an inherited abnormality or an acquired (condition that develops sometime later in life/after birth) abnormality; examples of abnormal small intestinal anatomy include partial blockages or obstructions of the small intestines; cancer; foreign body; folding of one segment of the intestine into another segment (known as “intussusception”); abnormal

narrowing of the small intestine (known as a “stricture”); presence of scar tissue that binds areas of the small intestines together (known as “adhesions”) and presence of a pouch or sac-like opening from the intestines (known as a “diverticulum”)

- Decreased intestinal motility (such as occurs with inadequate levels of thyroid hormone [known as “hypothyroidism”] and with disorders of the autonomic nervous system [known as “autonomic neuropathies”]); the “autonomic nervous system” is involved in the control of muscles in the heart, blood vessels, gastrointestinal tract, and other organs
- Exocrine pancreatic insufficiency (“EPI,” a syndrome caused by inadequate production and secretion of digestive enzymes by the pancreas)—approximately 70% of dogs with EPI have coexistent small intestinal bacterial overgrowth (SIBO)
- Abnormally low levels of hydrochloric acid in the stomach (known as “hypochlorhydria”) or lack of hydrochloric acid in the stomach (known as “achlorhydria”)—may occur spontaneously or may be related to medical treatment (such as treatment using H₂-blockers to decrease stomach acid)
- Inability to develop a normal immune response (known as “immunodeficiency”)
- Pre-existing intestinal disease

RISK FACTORS

- Suspected immunoglobulin A (IgA) deficiency; immunoglobulin A is an immune protein, found in the intestines; it functions as a protective barrier to prevent antigens (substances to which the immune system is responding and producing antibodies) and disease-causing microorganisms from entering the body through the intestines
- Intestinal disease (such as inflammatory bowel disease [IBD], adverse food reactions, parasite infestation) that affects local intestinal defense mechanisms



TREATMENT

HEALTH CARE

- Outpatient medical management
- Results/improvement may take a few days to several weeks
- Supportive care for emaciated pets or pets with low levels of albumin, a type of protein, in their blood (condition known as “hypoalbuminemia”)

ACTIVITY

- Unrestricted

DIET

- Highly digestible diet
- Diet containing fructo-oligosaccharides is the only diet that has been shown to be beneficial in dogs with small intestinal bacterial overgrowth (SIBO)

SURGERY

- Indicated for some underlying causes of small intestinal bacterial overgrowth (SIBO), such as partial blockages or obstructions of the intestines, tumors, or the presence of a pouch or sac-like opening from the intestines (diverticulum)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Broad-spectrum antibiotics administered by mouth; antibiotics effective against both bacteria that can live and grow in the presence of oxygen (known as “aerobic bacteria”) and bacteria that can live and grow in the absence of oxygen (known as “anaerobic bacteria”) are preferred
- Tylosin—primary antibiotic choice in the United States; usually a powder administered in the food; can be used long-term; for small dogs the drug should be reformulated into capsules; for larger dogs the dose can be approximated by using a teaspoon and administering the drug in food, as directed by your pet’s veterinarian
- Oxytetracycline—antibiotic with limited availability in the United States; do not administer with food (calcium in the diet binds with oxytetracycline and makes it ineffective)
- Metronidazole—antibiotic and antiprotozoal drug used to treat small intestinal bacterial overgrowth (SIBO) because of its activity

against anaerobic bacteria; also may have effects to modify the immune response (known as “immunomodulatory effects”); possibly useful in treating inflammatory bowel disease (IBD)

- Dogs with small intestinal bacterial overgrowth (SIBO) may be cobalamin (vitamin B₁₂) deficient; therefore, supplementation of vitamin B₁₂ by injection is indicated; blood tests to evaluate serum cobalamin and folate concentrations should be performed a month after the last dose of vitamin B₁₂
- Dogs with coexistent exocrine pancreatic insufficiency (“EPI;” a syndrome caused by inadequate production and secretion of digestive enzymes by the pancreas) and small intestinal bacterial overgrowth (SIBO)—treatment for SIBO is indicated only if pancreatic enzyme replacement therapy (for EPI) alone does not resolve the diarrhea and/or lead to weight gain



FOLLOW-UP CARE

PATIENT MONITORING

- Body weight
- In pets with low levels of albumin (a type of protein) in their blood (condition is hypoalbuminemia), blood work to evaluate serum albumin concentration
- Diarrhea also should resolve
- If diarrhea persists despite improved body weight and/or increased serum albumin concentration, diagnostic investigation for other intestinal disease is indicated

EXPECTED COURSE AND PROGNOSIS

- Primary small intestinal bacterial overgrowth (SIBO) without complicating factors (such as inflammatory bowel disease [IBD], intestinal cancer [lymphoma])—prognosis with appropriate antimicrobial therapy is usually good
- Prognosis for secondary SIBO depends on underlying disease



KEY POINTS

- Some pets show clinical improvement within days
- Some pets require weeks of therapy before demonstrating improvement—treat for 2 to 3 weeks before concluding that therapy is ineffective
- Any coexistent or predisposing diseases (such as partial blockage or obstruction of the intestines, intestinal cancer, exocrine pancreatic insufficiency [EPI], inflammatory bowel disease [IBD], or dietary intolerance/allergy) also must be treated
- Continual or repeated treatment often is required

SNEEZING, REVERSE SNEEZING, GAGGING



BASICS

OVERVIEW

- “Sneezing” is the forceful expelling of air (expiratory effort) through the nose, usually caused by irritation of the lining of the nose and nasal passages; it is a normal, protective reflex and commonly is associated with discharge from the nose
- “Reverse sneezing” is a sudden attack or spasm of noisy intake of air (inspiratory effort) to clear irritating materials or accumulated discharge from the back of the nasal passages; it is a normal, repetitive, protective reflex
- “Gagging” is a “heaving” or “choking” response due to contraction of the muscles of the throat; it is a normal, protective reflex to clear discharges from the voice box (larynx), upper windpipe (trachea), throat (pharynx) or esophagus (the tube running from the throat to the stomach); it is also called “retching” and is frequently misinterpreted as vomiting
- “Upper respiratory tract” or “upper airways” includes the nose, nasal passages, throat, and windpipe (trachea)
- “Lower respiratory tract” or “lower airways” includes the bronchi, bronchioles, and alveoli (the terminal portion of the airways, in which oxygen and carbon dioxide are exchanged)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Mean Age and Range

- Sneezing, reverse sneezing, and gagging are not associated with any specific age, but rather with conditions which cause them
- Young animals—examples include infections and cleft palate
- Older animals—examples include nasal tumors and dental disease

SIGNS/OBSERVED CHANGES in the ANIMAL

- Head and mouth position may help determine which of these reflexes is present
- Sneezing typically results in sudden, explosive expiratory effort(s), with the mouth closed and head thrown downward; may result in the animal’s nose hitting the ground
- Reverse sneezing is a sudden inspiratory effort, that frequently occurs as an “attack” or “spasm,” with the head pulled back, mouth closed and lips sucked in
- Gagging typically occurs with the head and neck extended, mouth held open and usually ends with the animal swallowing (with little to nothing expelled)
- Sneezing—often accompanied by discharge from the nose

CAUSES

- Any irritation or inflammation of the lining of the nose, voice box (larynx), or throat can elicit these reflexes; the same irritating agent in the nasal passages might elicit a sneeze, but when placed into the back of the throat would result in a reverse sneeze
- Common causes of sneezing and reverse sneezing include excess nasal discharge, foreign body (especially if signs are sudden [acute] and violent in onset), allergy, tumors and parasites (dogs—*Pneumonyssoides*; dogs and cats—*Cuterebra* and *Capillaria*; cats—*Linguatula*)
- Diseases outside of the nasal passages—pneumonia, enlarged esophagus (known as “megaesophagus”), long-term (chronic) vomiting, cricopharyngeal achalasia (a condition of the nerves and muscles in the upper esophagus, in which the muscles do not relax adequately to allow swallowing); discharge from these conditions may be forced up into the back of the throat, resulting in reverse sneezing, sneezing, and/or discharge from the nose
- Gagging is often due to discharges being coughed up from the lower airways and into the voice box (larynx) or upper windpipe (trachea); to dysfunction of the voice box (larynx) resulting in airway aspiration; or to vomiting from diseases of the esophagus (the tube running from the throat to the stomach) and gastrointestinal tract (stomach and intestines)

RISK FACTORS

- Poorly vaccinated animals may develop infection/inflammation of the upper airways (nose, nasal passages, and upper windpipe) and sneezing (for example, kittens with upper respiratory infections caused by viruses, puppies with kennel cough)
- Coughing may move discharges into the back of the throat and lead to reverse sneezing
- Long-term (chronic) dental disease may cause inflammation of the nose (known as “rhinitis”) and either sneezing or reverse sneezing
- Mites (type of parasite) in the nose may cause both sneezing and reverse sneezing in dogs (but not in cats)
- Foreign bodies in the nose will elicit sneezing and/or reverse sneezing, depending on their location; outdoor animals perhaps more at

risk



TREATMENT

HEALTH CARE

- Removal of the agent that is irritating the lining of the nose, voice box (larynx), or throat (where and when possible) will result in relief from these reflexes

SURGERY

- Depending on the underlying cause, anesthesia for surgery or use of a special lighted instrument called an “endoscope” (general term for procedure is “endoscopy”) may be needed to remove a foreign body causing discharge from the nose
- Anesthesia and dental surgery may be needed to remove an abscessed tooth
- Surgery may be necessary to treat disease of the voice box (larynx); however, risk of aspiration pneumonia should be considered when gagging is a prominent feature of the disease, due to an increased risk of aspiration pneumonia



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- No drug specifically suppresses these reflexes—treatment is directed at the underlying irritating agent
- Nasal bacterial infections (secondary to foreign body, dental disease, tumor) are best treated with antibiotics directed against gram-positive bacteria (most common)
- When no underlying nasal condition is found, long-term, non-specific treatment with doxycycline and piroxicam may be tried
- Nasal mites (type of parasite) are treated with ivermectin or milbemycin; all dogs in the household should be treated to prevent reinfection
- Lower airway diseases with excess discharge are treated with gram-negative spectrum antibiotics, if bacterial infection is confirmed
- For non-specific airway inflammation, use an anti-inflammatory (such as prednisolone or piroxicam), if no infection is confirmed
- Decongestants (such as ephedrine) or antihistamines may reduce discharges and sneezing in some cases



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on underlying cause

PREVENTIONS AND AVOIDANCE

- Depends on underlying cause
- Vaccinate against diseases that infect the upper respiratory tract (such as upper respiratory infections caused by viruses in kittens and cats, kennel cough in puppies and dogs)

POSSIBLE COMPLICATIONS

- Serious aspiration pneumonia may develop in some cases of gagging due to disease of the voice box (larynx)

EXPECTED COURSE AND PROGNOSIS

- Nasal mites (type of parasite) should respond within 3 weeks of treatment
- Gagging and reverse sneezing secondary to non-infectious causes resolve slowly



KEY POINTS

- Sneezing, reverse sneezing, and gagging are normal reflexes; diagnostics testing is required to determine the underlying cause and to allow appropriate treatment
- Close contact with other animals should be limited until treatment for the underlying cause is completed
- Episodes of paroxysmal reverse sneezing may be decreased by stimulating the dog to swallow (such as by rubbing the throat, giving water)

CONGENITAL SPINAL AND VERTEBRAL MALFORMATIONS



BASICS

OVERVIEW

- Abnormal development of spinal structures, which are apparent at birth or within the first weeks of life
- “Congenital” refers to something that is present at birth; “spinal” refers to the spine; “vertebral” refers to the vertebrae
- The spine is composed of multiple bones (vertebrae) with disks (intervertebral disks) located in between adjacent bones; the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae
- “Vertebra” (plural, vertebrae) is the medical term for backbone

GENETICS

- A genetic background, with unknown mode of inheritance, is suspected in most congenital (present at birth) diseases of the spine
- Defective development of the sacral and coccygeal (tail) vertebrae (known as “sacrococcygeal dysgenesis”)—autosomal dominant
- Incomplete development of one side of a thoracic vertebra (known as “thoracic hemivertebra”) of German shorthaired pointers—autosomal recessive

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Malformation of the bone at the back of the skull (known as the “occipital bone”), the first cervical vertebra (C₁; known as the “atlas”), and the second cervical vertebra (C₂; known as the “axis”)—most common in small-breed dogs
- Hemivertebra (incomplete development of one side of a vertebra), transitional vertebra (abnormal development of the backbone at the junction between two vertebral types, such as at the junction of the lumbar and sacral vertebrae), block vertebra (where two or more adjacent backbones are fused together), and butterfly vertebra (type of hemivertebra in which incomplete development of one side of the backbone causes a defect that looks like a butterfly on X-rays)—most common in short-nosed, flat-faced (known as “brachycephalic”), “screw-tailed” breeds (such as the French bulldog, English bulldog, pug, Boston terrier)
- Defective development of the sacral and coccygeal (tail) vertebrae (sacrococcygeal dysgenesis)—Manx cats
- Defective development of the spine leading to exposure of the covering of the spinal cord (known as “meninges”) or spinal cord (known as “spina bifida”)—bulldogs, Manx cats, and other screw-tailed breeds
- Abnormal development of the spinal cord (known as “myelodysplasia”)—Weimaraners
- Congenital (present at birth) narrowing of the spinal canal (known as “spinal stenosis”)—Doberman pinschers; breeds with “normal” short, bowed legs (known as “chondrodystrophic breeds”)

Mean Age Range

- Congenital (present at birth) malformations of the spine and/or backbones may cause clinical signs during the rapid growth of the animal (such as 5 to 9 months of age)
- Congenital (present at birth) spinal cord abnormalities cause clinical disease from the time of birth

SIGNS/OBSERVED CHANGES in the ANIMAL

- Distortion of the back or spine—lordosis (abnormal curvature that causes the spine to arch downwards, so called “sway back”); kyphosis (abnormal curvature that causes the spine to arch upwards); and scoliosis (abnormal curvature of the spine to one side) in cases of malformed backbones or vertebrae
- Incoordinated or “drunken” appearing gait or movement (known as “ataxia”) and weakness or partial paralysis (known as “paresis”) associated with squeezing or pressure on the spinal cord and trauma
- Signs vary with location of the spinal cord segment(s) involved

CAUSES

- Breed-related inherited defects are suspected for most congenital (present at birth) spinal abnormalities, although interactions between several genes and environmental factors (such as nutritional deficiencies) likely are involved

RISK FACTORS

- Compounds that cause abnormal development of the embryo (known as “teratogenic compounds”)
- Toxins
- Nutritional deficiencies
- Stress



TREATMENT

HEALTH CARE

- Depends on severity of nervous system deficits and related signs
- Outpatient—if animal is able to walk (known as being “ambulatory”)
- Inpatient—if animal is not able to walk (known as being “nonambulatory”) or requires emergency surgical treatment (such as for partial dislocation of the joint between the first and second cervical vertebra [condition known as “atlantoaxial subluxation”])
- Restricted activity combined with physical therapy—may help neurologically disabled patients in their postoperative period; a cart may be necessary for severely affected patients
- Management of urination may be essential for cases in which disorders of urination accompany the spinal abnormality

ACTIVITY

- Restricted, especially if partial dislocation of the vertebrae or backbones (known as “vertebral subluxation”) is present

SURGERY

- In general, surgery to relieve pressure on the spinal cord is required when congenital (present at birth) malformation(s) cause narrowing of the spinal canal and put pressure on the spinal cord; in cases of long-term (chronic) or widespread (diffuse) pressure on the spinal cord, therapeutic responses following surgery are minimal
- Partial dislocation of the joint between the first and second cervical vertebra (atlantoaxial subluxation)—surgery to relieve pressure on the spinal cord combined with stabilization of the atlantoaxial joint with pins or screws is the treatment of choice
- Defective development of the spine leading to exposure of the covering of the spinal cord (known as “meninges”) or spinal cord (spina bifida)—protrusion of the membranes around the spinal cord (meninges) through an opening in the spine (protruding membranes known as “meningoceles”) can be closed surgically to prevent leakage of cerebrospinal fluid and infections; surgery usually is not attempted when the spinal cord itself is involved



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids may be used in some cases; response to steroid treatment has been variable



FOLLOW-UP CARE

PATIENT MONITORING

- Frequent (such as every 4 to 6 months) nervous system examinations—often required to monitor the progression of clinical signs
- X-rays—repeat as needed

PREVENTIONS AND AVOIDANCE

- Avoid breeding affected animals

POSSIBLE COMPLICATIONS

- Depend on the type and severity of nervous system signs
- Partial dislocation of the joint between the first and second cervical vertebra (atlantoaxial subluxation)—sudden (acute) death may occur
- Partial dislocation of the vertebrae or backbones (vertebral subluxation)—sudden (acute) paralysis can be seen with further trauma and pressure on the spinal cord
- Surgical implant (such as bone plates or pins) failure may be observed after surgery to relieve pressure on the spinal cord and to stabilize the spine

EXPECTED COURSE AND PROGNOSIS

- Prognosis varies depending on the type of malformation, degree of pressure on the spinal cord or injury, and surgical techniques to relieve pressure on the spinal cord and to stabilize the spine
- Congenital (present at birth) malformation of vertebrae without pressure on the spinal cord—prognosis is good
- Partial dislocation of the joint between the first and second cervical vertebra (atlantoaxial subluxation) following surgery to relieve

pressure on the spinal cord and/or stabilization of the backbones—prognosis is fair to good

- Surgery to relieve pressure on the spinal cord—prognosis is fair
- Defective development of the spine leading to exposure of the covering of the spinal cord (known as “meninges”) or spinal cord (spina bifida) associated with spinal cord malformation; long-term (chronic) nervous system disease despite surgical treatment; and disease of the nerves that connect the spinal cord and muscles (known as “lower motor neuron disease”) with lack of control of urination (known as “incontinence”)—prognosis is poor
- Medical treatment usually is insufficient to alleviate moderate to severe nervous system signs caused by pressure on the spinal cord secondary to congenital (present at birth) malformation(s) of the vertebrae
- Many dogs and cats with nervous system signs that are not treated are euthanized



KEY POINTS

- Many congenital (present at birth) malformations of the vertebrae do not cause clinical signs
- A thorough diagnostic work-up should be performed when a congenial malformation results in nervous system abnormalities
- A genetic basis is suspected for malformations of the vertebrae; avoid breeding affected animals
- Early surgical intervention often is necessary to alleviate pressure on the spinal cord and to prevent further damage

SQUAMOUS CELL CARCINOMA OF THE SKIN

(A TYPE OF SKIN CANCER)



BASICS

OVERVIEW

- The “squamous epithelium” is the top layer of the skin, which is composed of flat, scale-like cells
- Squamous cell carcinoma is a cancer (malignant tumor) of squamous epithelium of the skin
- Bowen’s disease (cats)—multiple cancerous skin lesions, involving pigmented skin in areas with hair

GENETICS

- Unknown

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Cats—none reported; patients often have light or unpigmented skin
- Dogs—Scottish terriers, Pekingese, boxers, poodles, Norwegian elkhounds, Dalmatians, beagles, whippets, and white English bull terriers may be more likely than other dog breeds to develop squamous cell carcinoma of the skin; large-breed dogs with black skin and hair coats may be more likely to develop squamous cell carcinoma involving the digits

Mean Age and Range

- Dogs—9 years of age
- Cats—9 to 12.4 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Dried discharge on the surface of a skin lesion (known as a “crust”); superficial loss of tissue on the surface of the skin, frequently with inflammation (known as an “ulcer”); or mass that may have been present for months and unresponsive to conservative treatment
- Bowen’s disease (cats)—multiple cancerous skin lesions, involving pigmented skin in areas with hair; skin becomes pigmented; ulcer forms in the center; followed by a painful scabby lesion that may expand peripherally
- Lips, nose, and ear (pinna) involvement—may start out as a shallow crusting lesion that progresses to a deep ulcer
- Facial skin involvement (cats)
- Nail-bed involvement (dogs)

Physical Examination Findings

- Rapidly growing lesion (known as a “proliferative lesion”) or lesions with loss of surface tissue (erosive lesions)
- Most common sites—cats: the tough, hairless skin of the nose (known as the “nasal planum”), eyelids, lips, and ear (pinna); dogs: toes, scrotum, nose, legs, and anus
- Skin on the flank and/or abdomen may be involved
- Bowen’s disease (cats)—may note 2 to more than 30 cancerous lesions on the head, digits, neck, chest, shoulders, and lower abdomen; hair in the lesion pulls out easily; crusts (dried discharge) cling to the hairs

CAUSES

- Unknown
- Exposure to ultraviolet irradiation

RISK FACTORS

- Sunny climates and high altitudes (high ultraviolet light exposure)
- Prolonged exposure to ultraviolet light
- Light or nonpigmented skin
- Previous thermal injury—burn scar



TREATMENT

HEALTH CARE

- Invasive tumors are tumors that extend into deeper tissues (that is, they involve more than the surface of the skin)—inpatient; require aggressive surgical excision or radiation therapy
- Superficial tumors—surgical removal; freezing the tissue to destroy it (known as “cryosurgery”); using light energy to destroy tissue (known as “photodynamic therapy”); or radiation treatment
- Topical synthetic retinoids—retinoids are chemicals with vitamin A activity; may be useful for early superficial lesions

ACTIVITY

- Dictated by the location of the tumor and the type of treatment
- Generally limited activity, until sutures are removed, if surgery has been done

DIET

- Normal
- Feeding tube may be necessary with surgical removal of cancer involving the tough, hairless skin of the nose (nasal planum)

SURGERY

- Wide surgical excision (that is, surgically removing the tumor and wide borders of apparently normal tissue)—treatment of choice; skin flaps and body wall reconstruction sometimes required
- Digit involvement—amputation
- Ear (pinna) involvement—may require partial or complete surgical removal of the ear
- Tumors that extend into deeper tissues of the nostrils—surgical removal of the tough, hairless skin of the nose (nasal planum) is recommended
- Radiation therapy—recommended for inoperable tumors (that is, tumors that cannot be removed surgically) or in addition to surgery



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Chemotherapy—recommended in cases with surgery in which cancer or cancer cells remain (known as “incomplete surgical excision”); with inoperable tumors (that is, tumors that cannot be removed surgically); and with spread of the cancer to other parts of the body (known as “metastasis”)—cisplatin (dogs), carboplatin, and mitoxantrone—reported to induce partial and complete remission; generally remission is of short duration
- Topical synthetic retinoids—retinoids are chemicals with vitamin A activity; may be useful for early superficial lesions



FOLLOW-UP CARE

PATIENT MONITORING

- Physical examination and X-rays—1, 3, 6, 9, 12, 18, 21, and 24 months after treatment or if the owner thinks the tumor is recurring
- Chest X-rays at each recheck examination to evaluate possible spread of the cancer (metastasis) into the lungs; abdominal X-rays or ultrasound, determined by location of the cancer

PREVENTIONS AND AVOIDANCE

- Limit sun exposure, especially between the hours of 10:00 a.m. and 2:00 p.m.
- Yearly tattoos on nonpigmented areas may be helpful
- Sunscreens—usually licked off by the patient; may help in some areas (such as the ear [pinna])

EXPECTED COURSE AND PROGNOSIS

●Prognosis—good with superficial lesions that receive appropriate treatment; guarded with invasive tumors that extend into deeper tissues (that is, they involve more than the surface of the skin) and those involving the nail bed or digit



KEY POINTS

- Early diagnosis and treatment for squamous cell carcinoma of the skin is important
- Risk factors associated with the development of the tumor include sunny climates and high altitudes (high ultraviolet light exposure)
- Limit sun exposure, especially between the hours of 10:00 a.m. and 2:00 p.m.
- Prognosis—good with superficial lesions that receive appropriate treatment; guarded with invasive tumors that extend into deeper tissues (that is, they involve more than the surface of the skin) and those involving the nail bed or digit

NOISY BREATHING (STERTOR AND STRIDOR)



BASICS

OVERVIEW

- Unusually loud breathing sounds that result from air passing through abnormal narrowings involving the back of the throat (known as the “nasopharynx”), the throat (known as the “pharynx”), the voice box (known as the “larynx”), or the windpipe (known as the “trachea”) and meeting resistance to airflow because of partial blockage of these regions
- Abnormal breathing sounds can be heard without using a stethoscope
- “Stertor” is noisy breathing when inhaling; it is a low-pitched, snoring sound that usually arises from the vibration of relaxed or flabby tissue or fluid; usually arises from airway blockage in the throat (pharynx)
- “Stridor” is high-pitched, noisy breathing; the higher-pitched sounds result when relatively rigid tissues vibrate with the passage of air; result of partial or complete blockage of the nasal passages or voice box (larynx) or collapse of the upper part of the windpipe (known as “cervical tracheal collapse”)
- “Upper respiratory tract” or “upper airways” includes the nose, nasal passages, throat (pharynx), and windpipe (trachea)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Common in short-nosed, flat-faced (known as “brachycephalic”) dogs or cats
- Inherited paralysis of the voice box (known as “laryngeal paralysis”)—identified in Bouviers des Flandres, Siberian huskies, bulldogs, and Dalmatians
- Acquired (condition that develops sometime later in life/after birth) paralysis of the voice box (laryngeal paralysis)—more common in certain giant-breed dogs (such as St. Bernards and Newfoundlands) and large-breed dogs (such as Irish setters, Labrador retrievers, and golden retrievers) than other breeds

Mean Age and Range

- Affected short-nosed, flat-faced (brachycephalic) animals and dogs or cats with inherited paralysis of the voice box (laryngeal paralysis) typically are younger than 1 year of age when breathing problems are detected
- Acquired (condition that develops sometime later in life/after birth) paralysis of the voice box (laryngeal paralysis) typically occurs in older dogs and cats
- Cats—diagnosed less commonly than are dogs; no obvious age pattern

Predominant Sex

- Inherited paralysis of the voice box (laryngeal paralysis) has a 3:1 male-to-female ratio

SIGNS/OBSERVED CHANGES in the ANIMAL

- Change or loss of voice
- Partial blockage of the upper airways produces an increase in airway sounds, before producing an obvious change in breathing pattern
- Unusually loud breathing sounds may have existed for as long as several years
- Breath sounds can be heard from a distance, without the use of a stethoscope
- Nature of the sound—ranges from abnormally loud to obvious fluttering to high-pitched squeaking, depending on the degree of airway narrowing
- May note increased breathing effort; breathing often accompanied by obvious body changes (such as extended head and neck and open-mouth breathing)

CAUSES

- Condition of abnormal breathing passages in short-nosed, flat-faced animals (condition known as “brachycephalic airway syndrome”), characterized by any combination of the following conditions: narrowed nostrils (known as “stenotic nares”); overly long soft palate; turning inside-out of a portion of the voice box or larynx (known as “everted laryngeal sacculles”), such that the space for air to pass through the larynx is decreased; and collapse of the voice box or larynx (known as “laryngeal collapse”), and fluid-build up (known as “edema”) of the voice box or larynx
- Narrowing of the back of the nose and throat (known as “nasopharyngeal stenosis”)
- Paralysis of the voice box or larynx (laryngeal paralysis)—inherited or acquired (condition that develops sometime later in life/after birth)

- Tumors of the voice box or larynx—benign or malignant (cancer)
- Nodular, inflammatory lesions of the voice box or larynx (known as “granulomatous laryngitis”)
- Reduction in the diameter of the lumen of the windpipe (trachea) during breathing (known as “tracheal collapse”)
- Narrowing of the windpipe (trachea; condition known as “tracheal stenosis”)
- Tumors of the windpipe (trachea)
- Foreign bodies in the windpipe (trachea) or other parts of the airway
- Inflammatory masses that develop from the middle ear or eustachian tube (known as “nasopharyngeal polyps”)
- Condition caused by excessive levels of growth hormone, leading to enlargement of bone and soft-tissues in the body (known as “acromegaly”)
- Nervous system and/or muscular dysfunction
- Anesthesia or sedation—if certain anatomy exists (such as a long soft palate) that increases susceptibility to abnormal, loud breathing sounds
- Abnormalities or tumors of the soft palate (the soft portion of the roof of the mouth, located between the hard palate and the throat)
- Excessive lining tissue of the throat (known as “redundant pharyngeal mucosal fold”)
- Tumor in the back of the throat (pharynx)
- Fluid build-up (edema) or inflammation of the palate, throat (pharynx), and voice box (larynx)—secondary to coughing, vomiting or regurgitation, turbulent airflow, upper respiratory infection, and bleeding
- Discharges (such as pus, mucus, and blood) in the airway lumen—suddenly (acutely) after surgery; a normal conscious animal would cough out or swallow them

RISK FACTORS

- High environmental temperature
- Fever
- High metabolic rate—as occurs with increased levels of thyroid hormone (known as “hyperthyroidism”) or a generalized bacterial infection (known as “sepsis”)
- Exercise
- Anxiety or excitement
- Any breathing or heart disease that increases movement of air into and out of the lungs (known as “ventilation”)
- Turbulence caused by the increased airflow may lead to swelling and worsen the airway obstruction
- Eating or drinking



TREATMENT

HEALTH CARE

- Keep patient cool, quiet, and calm—anxiety, exertion, and pain lead to increased movement of air into and out of the lungs (ventilation), potentially worsening the blockage to airflow
- Low levels of oxygen in the blood and tissues (known as “hypoxia”) and decreased movement of air into and out of the lungs (known as “hypoventilation”) occur with prolonged, severe blockage to airflow; supplemental oxygen not always critical for sustaining patients with partial airway collapse
- Closely monitor effects of sedatives; sedatives may relax the upper airway muscles and worsen the blockage to airflow; be prepared for emergency treatment if complete obstruction occurs

ACTIVITY

- Activity determined by underlying cause
- Exercise is a risk factor; therefore, limited exercise may be necessary, as directed by your pet’s veterinarian

SURGERY

- Extreme airway blockage or obstruction—attempt an emergency intubation (that is, passage of an endotracheal tube through the mouth and into the windpipe [trachea] to allow oxygen to reach the lungs); if obstruction prevents intubation, emergency tracheotomy (surgical opening into the windpipe [trachea]) or passage of a tracheal catheter to administer oxygen may be the only available means for sustaining life; a tracheal catheter can sustain oxygenation only briefly while a more permanent solution is sought
- Utilization of small balloon catheters may be useful in dislodging foreign bodies
- Surgery—biopsy to determine type of mass in the airways; surgical treatment (such as surgical removal of mass, correction of airway

defects, or removal of foreign bodies)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Medical approaches—appropriate only if the underlying cause is infection, fluid build-up (edema), inflammation, or bleeding; structural/anatomic abnormalities or nervous system causes are not responsive to symptomatic medical treatment
- Steroids—may be indicated if fluid build-up (edema) or inflammation is thought to be an important contributor to the abnormal, loud breathing sounds



FOLLOW-UP CARE

PATIENT MONITORING

- Breathing rate and effort need to be monitored closely—complete blockage or obstruction could occur when an apparently stable patient is taken home or if continual observation is not feasible

PREVENTIONS AND AVOIDANCE

- Avoid exercise, high ambient temperatures, and extreme excitement

POSSIBLE COMPLICATIONS

- Serious complications may occur without treatment to relieve the airway blockage or obstruction; these include fluid build-up in the airways (airway edema) and/or lungs (known as “pulmonary edema,” which may progress to life-threatening lung injury), and decreased movement of air into and out of the lungs (hypoventilation); may require tracheotomy (surgical opening into the windpipe [trachea]) and/or artificial ventilation (such as use of a mechanical respirator)
- Particular care should be taken when inducing general anesthesia or when using sedatives in any patient with upper airway blockage or obstruction
- The patient can transition from being a noisy breather to having a blocked or obstructed airway in a few minutes or even seconds

EXPECTED COURSE AND PROGNOSIS

- Varies with underlying cause
- Even with surgical treatment, some degree of obstruction may remain for 7 to 10 days due to postoperative swelling



KEY POINTS

- “Stertor” is noisy breathing when inhaling; it is a low-pitched, snoring sound that usually arises from the vibration of relaxed or flabby tissue or fluid; usually arises from airway blockage in the throat (pharynx)
- “Stridor” is high-pitched, noisy breathing; the higher-pitched sounds result when relatively rigid tissues vibrate with the passage of air; result of partial or complete blockage of the nasal passages or voice box (larynx) or collapse of the upper part of the windpipe (known as “cervical tracheal collapse”)
- The patient can transition from being a noisy breather to having a blocked or obstructed airway in a few minutes or even seconds
- Serious complications may occur without treatment to relieve the airway blockage or obstruction

STOMATITIS

(INFLAMMATION OF THE MOUTH)



BASICS

OVERVIEW

- “Stomatitis” is inflammation of the lining tissues (known as “mucous membranes”) of the mouth
- The inflammation involves the soft tissues (such as gums and tongue) of the mouth
- Inflammation may be caused by many different stimuli of local (that is, within the mouth itself) or generalized (systemic) origin

GENETICS

- Oral eosinophilic granuloma (a mass or nodular lesion located in the mouth, containing a type of white-blood cell, called an eosinophil)—most commonly seen in the Siberian husky (may be hereditary)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dog and cat

Breed Predilections

- Ulcerative stomatitis in Maltese (dogs); ulcerative stomatitis is a condition in which the gum tissue is very fragile and is characterized by significant loss of surface gum tissue, frequently with inflammation (known as “ulcers”)
- Oral eosinophilic granuloma (a mass or nodular lesion located in the mouth, containing a type of white-blood cell, called an eosinophil)—most commonly seen in the Siberian husky (may be hereditary)
- Gingival hyperplasia in large-breed dogs; gingival hyperplasia is a condition in which the gum tissue increases in size, leading to thickened, enlarged gums
- Rapidly progressive inflammation/infection of the gums and supporting tissues of the teeth (known as “periodontitis”) seen mostly in young adult animals, such as the greyhound and the shih tzu
- Lymphocytic plasmacytic stomatitis in cats; “lymphocytic plasmacytic stomatitis” is inflammation of the lining of the mouth, characterized by the presence of lymphocytes and plasma cells; lymphocytes are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process; plasma cells or plasmacytes are a specialized type of white-blood cell; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease
- Localized inflammation/infection of the gums and supporting tissues of the teeth in young animals (condition known as “juvenile periodontitis”) in the incisor region of the upper jaw (maxilla) or lower jaw (mandible)—especially common in the miniature schnauzer

Mean Age and Range

- Juvenile-onset periodontitis in the miniature schnauzer and in young cats
- Periodontal disease associated with calculus is seen most often in older dogs and cats

Predominant Sex

- Ulcerative stomatitis in Maltese—higher incidence in male dogs

SIGNS/OBSERVED CHANGES in the ANIMAL

- Bad breath (known as “halitosis”)
- Pain
- Ulcerated lesions
- Excessive salivation/drooling (known as “ptyalism”)
- Fluid-build up in the soft tissues, such as the gums (fluid build-up known as “edema”)
- Skin problems (such as draining lesions, redness, swelling) around the eye area (known as “periocular inflammation”) is possible, due to anatomic relationship between the teeth, sinuses, and the area near the eye
- Extensive plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva) and tartar or calculus (mineralized plaque on the tooth surface); ulcerated lesions may be seen on the surfaces of the lining of the mouth (for example, the lining of the cheeks) that are adjacent to teeth with large amounts of calculus

CAUSES

Anatomic (Structural) Disorders

- Inflammation/infection of the gums and supporting structures of the teeth (periodontal disease) due to overcrowding of teeth

- Attachment of the fold of tissue extending from the gum to the lip (located at the midline of the gum; condition known as a “lip frenulum attachment”)
- Tight-lip syndrome in the Chinese shar pei; “tight-lip syndrome” is a condition in which the lower lip is pulled up tightly against the lower incisors

Metabolic Disorders

- Excess levels of urea and other nitrogenous waste products (known as “uremia”) and high ammonia levels in saliva
- Inflammation of blood vessels (known as “vasculitis”) and dry mouth (known as “xerostomia”) seen with sugar diabetes (diabetes mellitus)
- Enlargement of the tongue (known as “macroglossia”) and puffy lips, as seen with inadequate levels of parathyroid hormone produced by the parathyroid glands (condition known as “hypoparathyroidism”)
- Lymphoma can be seen affecting the palate and/or tongue; lymphoma is a type of cancer that develops from lymphoid tissue, including lymphocytes, a type of white-blood cell formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process

Immune-Mediated Diseases

- Pemphigus foliaceus
- Pemphigus vulgaris
- Bullous pemphigoid
- Systemic lupus erythematosus and discoid lupus erythematosus in the dog
- Sudden (acute) allergic reaction (hypersensitivity) to drugs

Infectious Disease

- Normal bacteria in the mouth can cause infection if the lining of the mouth is disrupted (for example, injured by a foreign body)
- Fungal infection of the mouth (known as “mycotic stomatitis”)
- Generalized (systemic) infections
- Leptospirosis can cause pinpoint bruises (known as “petechia”) in the mouth
- Feline leprosy (*Mycobacterium* infection) can cause raised patches (known as “plaques”) in the mouth
- Calicivirus or herpesvirus infections—cat
- Canine distemper virus—dogs
- Wart-like lesions in the mouth caused by a viral infection (known as “viral papillomatosis”)—dogs

Trauma

- Irritation from plaque (the thin, “sticky” film that builds up on the teeth; composed of bacteria, white-blood cells, food particles, and components of saliva) and tartar or calculus (mineralized plaque on the tooth surface)
- Foreign objects
- “Gum-chewer’s disease”—chronic chewing of the moist tissues lining the cheek
- Electrical cord shock
- Chemical burns
- Lacerations
- Snake bite
- Blows
- Trauma of the palate from contact with the canine teeth of the lower jaw (mandibular canine teeth) that are too close together, so they do not fit in normal location when the mouth closes

Toxic Injury

- Certain plants
- Chemotherapy
- Radiation therapy
- Chemical irritants



TREATMENT

HEALTH CARE

- Dental disease or inflammation/infection of the gums and supporting tissues of the teeth (periodontal disease) should be treated
- Treatment of lymphoma (cancer of lymphoid tissue), if causing stomatitis

DIET

- Correct nutritional or hydration deficiencies, as needed; on an inpatient or outpatient basis
- Can use feeding tube, if necessary

SURGERY

- Most or all teeth must be extracted to resolve inflammation of the mouth (stomatitis) in many cases



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Broad-spectrum antibiotics—amoxicillin-clavulanate; clindamycin; metronidazole; doxycycline
- Products applied directly to the mouth (known as “topical products”)—chlorhexidine solution or gel (CHX™, VRx Products, Harbor City, CA) is a plaque retardant; Maxi/Guard® (Addison Biological Laboratory, Fayette, MO) zinc-organic acid solutions and gels to promote tissue healing and retard plaque accumulation
- Anti-inflammatory drugs, such as prednisolone or prednisone; may be used for treatment of inflammation of the gums and throat characterized by the presence of plasma cells (a specialized type of white-blood cell; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease) in cats; the disorder is known as “feline plasma-cell gingivitis-pharyngitis;” may improve inflammation and appetite



FOLLOW-UP CARE

PATIENT MONITORING

- Laboratory tests, when generalized (systemic) disease is involved
- Oral rinses and brushing the teeth may be helpful; frequently used in a dental home-care program, especially for inflammation/infection of the gums and supporting tissues of the teeth (periodontal disease)

PREVENTIONS AND AVOIDANCE

- OraVet™ (Merial, Atlanta, GA) applied weekly to calculus-free teeth may be helpful in preventing further inflammation of the mouth
- Oral rinses and brushing the teeth may be helpful, especially for inflammation/infection of the gums and supporting tissues of the teeth (periodontal disease)
- A periodontal vaccine (Pfizer) has been developed to aid in the prevention of periodontal disease

POSSIBLE COMPLICATIONS

- Bacteria involved with inflammation/infection of the gums and supporting tissues of the teeth (periodontal disease) can enter the blood stream (known as “bacteremia”) and can cause kidney, heart, liver, and lung disease

EXPECTED COURSE AND PROGNOSIS

- Depends on disease causing the inflammation of the lining of the mouth (stomatitis)



KEY POINTS

- “Stomatitis” is inflammation of the lining tissues (known as “mucous membranes”) of the mouth
- Inflammation may be caused by many different stimuli of local (that is, within the mouth itself) or generalized (systemic) origin
- Bacteria involved with inflammation/infection of the gums and supporting tissues of the teeth (periodontal disease) can enter the blood stream (known as “bacteremia”) and can cause kidney, heart, liver, and lung disease
- Oral rinses and brushing the teeth may be helpful, especially for inflammation/infection of the gums and supporting tissues of the teeth (periodontal disease)

STUPOR AND COMA



BASICS

OVERVIEW

- “Stupor” is the medical term for impaired consciousness, in which the animal must be stimulated to be awakened
- “Coma” is the medical term for unconsciousness or lack of consciousness, in which the animal cannot be stimulated to be awakened

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- The pet may have had trauma or unsupervised roaming, or may have medical problems of significance (such as diabetes mellitus, low blood sugar [known as “hypoglycemia”], heart problems, kidney failure, liver failure, or cancer) that cause various signs other than stupor or coma
- Onset of stupor or coma may be sudden (acute) or slowly progressive, depending on underlying cause
- Severely low body temperature (known as “severe hypothermia”) or severely high body temperature (known as “severe hyperthermia”)
- Evidence of bleeding—small, pinpoint areas of bleeding (known as “petechiae”); bruises or purplish patches under the skin, due to bleeding (known as “ecchymoses”)
- Bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red-blood cells (condition known as “cyanosis”)
- Heart or breathing abnormalities
- High blood pressure (known as “hypertension”)
- Nervous system abnormalities (such as abnormal pupils or short, rapid movements of the eyeball [known as “nystagmus”])

CAUSES

- Drugs—narcotics; depressants; ivermectin
- Structural or anatomic abnormalities—hydrocephalus (condition characterized by fluid build-up in the brain; also known as “water on the brain”)
- Metabolic abnormalities—numerous abnormalities, such as severely low blood sugar (severe hypoglycemia); high blood sugar (known as “hyperglycemia”); high levels of sodium in the blood (known as “hypernatremia”); low levels of sodium in the blood (known as “hyponatremia”); nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”); low levels of oxygen in the blood (known as “hypoxemia”); increased levels of carbon dioxide in the blood (known as “hypercarbia”); low body temperature (hypothermia); high body temperature (hyperthermia); low blood pressure (known as “hypotension”); blood-clotting disorders (known as “coagulopathies”); kidney failure
- Nutritional disorders—low blood sugar (hypoglycemia); thiamine (vitamin B1) deficiency
- Primary tumors or cancer involving the brain—meningioma; astrocytoma; gliomas; choroid plexus papilloma; pituitary adenoma; others
- Cancer that has spread to the brain (known as “metastatic cancer”)—hemangiosarcoma; lymphoma; breast cancer (known as “mammary carcinoma”); others
- Inflammatory noninfectious disease—granulomatous meningoencephalomyelitis (nodular inflammation of the brain, spinal cord and their surrounding membranes [the membranes are known as “meninges”])
- Infectious diseases—bacterial infections; viral infections (such as canine distemper, feline infectious peritonitis [FIP]); parasitic infections; protozoal infections (such as neosporosis, toxoplasmosis); fungal infections (such as cryptococcosis, blastomycosis, histoplasmosis, coccidioidomycosis, actinomycosis); rickettsial infections
- Unknown cause (so called “idiopathic disease”)—epilepsy
- Immune-mediated disease—inflammation of blood vessels (known as “vasculitis”) and low platelet count (known as “thrombocytopenia”) leading to bleeding; “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- Trauma
- Poisons—ethylene glycol (found in antifreeze); lead; rodenticide anticoagulants; others

- Vascular—bleeding disorders, high blood pressure (hypertension); sudden lack of blood supply that leads to death of tissues (known as “infarction”), such as seen in feline ischemic encephalopathy or migrating adult heartworm in the brain

RISK FACTORS

- Diabetes mellitus (“sugar diabetes”)—insulin therapy
- Tumor of the pancreas involving the cells that secrete insulin (known as an “insulinoma”)
- Severe heat or cold exposure without protection to maintain body temperature within normal limits
- Free-roaming animals—trauma
- Young and unvaccinated animals



TREATMENT

HEALTH CARE

- Maintain blood pressure; avoid high blood pressure (hypertension)
- Maintain hydration—cautious use of fluids, as they can contribute to fluid build-up in the brain (known as “cerebral edema”)
- The head should be level with the body or elevated to a 20° angle; the head should never be lower than the body to avoid increase in pressure within the skull/brain (known as “intracranial pressure”)
- Prevent thrashing, seizures, or any other form of uncontrolled motor activity; may elevate pressure within the skull/brain (intracranial pressure); medications to cause relaxation or to control seizures (such as diazepam) may be required
- Meticulous nursing care to prevent secondary complications—eye lubrication to keep the eyes moist; sterile technique with placement of catheters; turning periodically to avoid lung congestion and development of “bed sores” (known as “decubital ulcers”)
- Drainage of cerebrospinal fluid (CSF) in some cases, if critical elevation of pressure within the skull/brain (intracranial pressure) does not respond to medical treatment

DIET

- Nutrition—maintain during the unconscious period with nutrients administered through feeding tubes or by intravenous routes; adjust nutritional requirements

SURGERY

- Surgical decompression and exploration—seriously consider if nervous system signs worsen, including progressive brain dysfunction with a history of trauma or bleeding, high pressure within the skull/brain (intracranial pressure) that is not responsive to medical therapy, depressed skull-fracture fragments, and penetrating foreign body



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

High Pressure within the Skull/Brain (Intracranial Pressure)

- Increased ventilation to remove excess carbon dioxide from the body (known as “hyperventilation”) or medication to remove excess fluid from the brain (known as a “diuretic”), such as furosemide or mannitol
- Consider medication to control seizures (such as phenobarbital), if on-going seizure activity suspected

Underlying Disease

- Steroids—to treat inflammatory and space-occupying abnormalities within the skull/brain
- Lactulose enemas and fluid support—to treat hepatic encephalopathy (nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia)
- Increase urine production by administration of fluids (known as “fluid diuresis”)—to treat kidney failure
- Rehydration and insulin—to treat diabetes mellitus (“sugar diabetes”; lower blood sugar (glucose) slowly
- Glucose supplementation—to treat low blood sugar (hypoglycemia)
- Fluid support of blood volume; cool body temperature—to treat high body temperature (hyperthermia)
- Fluid support of blood volume; warm body temperature—to treat low body temperature (hypothermia)
- Flush out the stomach (known as “gastric lavage”) and instill activated charcoal—to treat suspected ingestion of a poison (treatment

determined by type of poison)

- Specific poisons may require specific treatments (such as administration of an antidote)
- Antibiotics—examples include trimethoprim-sulfa, chloramphenicol, and metronidazole, and first-generation cephalosporins
- Adjust fluid selection to correct electrolyte disorders.
- Drugs that improve the propulsion of contents through the stomach and intestines (known as “gastrointestinal prokinetic agents” or “motility modifiers”), such as cisapride, may be necessary



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat nervous system examinations—detect deterioration of function that warrants aggressive therapeutic intervention
- Blood pressure—keep fluid therapy adequate for normal blood flow, while avoiding high blood pressure (hypertension)
- Blood gases (measurements of oxygen and carbon dioxide levels in arterial blood)—assess need for oxygen supplementation or ventilation; monitor carbon dioxide levels when hyperventilating
- Blood glucose—ensure adequate blood level to maintain brain functions
- Electrocardiogram (“ECG,” a recording of the electrical activity of the heart)—detect irregular heart beats (known as “arrhythmias”) that may affect blood flow and oxygen levels
- Monitor pressure within the skull/brain (intracranial pressure), if have necessary monitoring instrumentation—detect marked elevations; track success of treatment
- Electrolytes—detect increased levels of sodium in the blood (hypernatremia) and decreased levels of potassium in the blood (known as “hypokalemia”)

PREVENTIONS AND AVOIDANCE

- Keep pets confined or leashed
- Prevent exposure to poisons or in-home medications
- Routine health-care program to minimize infectious and metabolic disease complications

POSSIBLE COMPLICATIONS

- Residual nervous system deficits, such as gait abnormalities or seizures
- Complications consistent with underlying disease

EXPECTED COURSE AND PROGNOSIS

- Vary with underlying cause
- Prognosis is worse with cases of brain-stem disease than with disease of the cerebral cortex (the outer layer of the brain)

RAPID HEART RATE: SUPRAVENTRICULAR TACHYCARDIA



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- The normal heart rate for dogs varies based on the size of the dog; however, the general range is 60 to 180 beats per minute (with smaller dogs have faster normal heart rates)
- The general range for normal heart rate in cats is 120 to 240 beats per minute
- “Supraventricular” refers to “above the ventricles,” and usually is used to indicate a heart rhythm that is started in tissues above the ventricles; “tachycardia” is a fast or rapid heart rate
- “Supraventricular tachycardia” is a rapid heart rate caused by electrical impulses that originate from a site other than the sinoatrial (SA) node, such as the muscle of the atria (known as “atrial myocardium”) or the atrioventricular (AV) node

GENETICS

- A genetic basis is suspected in Labrador retrievers

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and rarely cats

Breed Predilections

- Labrador retrievers

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs may relate to the underlying cause
- Some dogs may exhibit no clinical signs; rapid heart rate detected on physical examination
- Dogs with fast supraventricular tachycardia (heart rate usually greater than 300 beats per minute) generally exhibit episodic weakness or fainting (known as “syncope”)
- Coughing or breathing abnormalities in dogs with congestive heart failure (“CHF”); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Rapid, usually regular heart rate
- May have evidence of poor circulation—pale gums and moist tissues of the body (known as “mucous membranes”); the pink color of the gums is slow to return when the gums are blanched by finger pressure (known as “prolonged capillary refill time”); and weak pulses
- Underlying heart condition (such as a heart murmur)

CAUSES

- Long-term (chronic) disease of the heart valves
- Disease of the heart muscle (known as “cardiomyopathy”)
- Congenital (present at birth) heart disease
- Heart tumors or cancer
- Generalized (systemic) disorders
- Condition in which a pathway (known as an “accessory pathway”) to transmit the electrical impulse from the atria to the ventricles bypasses the atrioventricular (AV) node, and allows the impulse to reach the ventricles too quickly (known as “ventricular preexcitation”)
- Electrolyte imbalances

- Digoxin toxicity; digoxin is a heart medication
- Unknown cause (so called “idiopathic disease”)

RISK FACTORS

- Heart disease
- Genetics in Labrador retrievers



TREATMENT

HEALTH CARE

- Patients without clinical signs can be managed on an outpatient basis
- Patients with sustained, rapid heart rates (supraventricular tachycardia) or signs of congestive heart failure (CHF) should be hospitalized until stable; CHF signs include cough; difficulty breathing (dyspnea); bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Rapid heart rate (supraventricular tachycardia) is a medical emergency in dogs that exhibit weakness and collapse
- Electrical shock to the heart (known as “cardioversion”) or a pacemaker may be considered in extreme cases
- Treat congestive heart failure and correct any underlying electrolyte or acid–base disturbances (abnormalities in blood pH levels)

ACTIVITY

- Restrict until the rapid heart rate (supraventricular tachycardia) has been controlled

DIET

- Mild to moderate sodium restriction if patient is in congestive heart failure; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

SURGERY

- Consider procedure (known as “transvenous catheter ablation”) for patients with accessory pathways



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Emergency Treatment

- Administer one of the following drugs: calcium channel blocker (verapamil or diltiazem) or β -adrenergic blocker (esmolol)
- Intravenous (IV) adenosine (adenosine is very expensive and has a short duration of action); propranolol (propranolol has a longer duration of action after IV administration and generally is not recommended, unless no other alternative is available)

Long-term Treatment

- Digoxin—a heart medication
- β -adrenergic blocker—atenolol can be administered as long as the patient does not have underlying moderate-to-severe heart muscle failure (known as “myocardial failure”)
- Diltiazem is the calcium channel blocker of choice for long-term control of rapid heart rate (supraventricular tachycardia)
- Medications to control irregular heart beats (known as “antiarrhythmic agents,” such as quinidine and procainamide) can be tried



FOLLOW-UP CARE

PATIENT MONITORING

- Serial recordings of an electrocardiogram (“ECG,” a recording of the electrical activity of the heart) or Holter monitoring (where the patient wears a “vest” in which a continuous, mobile battery-powered ECG monitor has been placed; the ECG recording is performed over several hours, giving a better overall picture of the heart rate and rhythm)

POSSIBLE COMPLICATIONS

- Fainting (syncope) and congestive heart failure; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

EXPECTED COURSE AND PROGNOSIS

- Most cases are controlled effectively with medication



KEY POINTS

- Observe patients closely for signs of low blood volume being pumped by the heart (known as “low cardiac output”), such as weakness and collapse
- Rapid heart rate (supraventricular tachycardia) is a medical emergency in dogs that exhibit weakness and collapse

FAINTING (SYNCOPE)



BASICS

OVERVIEW

- Temporary loss of consciousness due to decreased oxygen reaching the brain, usually associated with abnormal circulation of blood, with spontaneous recovery
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Sick sinus syndrome (condition in which the normal “pacemaker” function of the sinoatrial (SA) node of the heart is abnormal and may lead to the heart rate being alternately slow and fast)—cocker spaniel, miniature schnauzer, pug, dachshund
- Ventricular irregular heart beats (known as “ventricular arrhythmias”)—boxer, German shepherd dog

Mean Age and Range

- More common in old animals

SIGNS/OBSERVED CHANGES in the ANIMAL

- Depend on underlying cause
- Sudden loss of consciousness that resolves quickly
- Collapse

CAUSES

Heart Causes

- Slow heart rate (known as “bradycardia” or “bradyarrhythmia”)—types include sinus bradycardia, sinus arrest, second-degree heart or atrioventricular (AV) block, complete heart or AV block, atrial standstill
- Rapid heart rate (known as “tachycardia” or “tachyarrhythmia”)—types include ventricular tachycardia, supraventricular tachycardia, atrial fibrillation
- Low blood volume being pumped by the heart (known as “low cardiac output”) not related to irregular heart beats (arrhythmias)—disease of heart muscle (known as “cardiomyopathy”); long-term (chronic) mitral valve disease; birth defect involving narrowing just below the aortic valve, the heart valve from the left ventricle to the aorta (the main artery of the body; condition known as “subaortic stenosis”); birth defect involving narrowing of the valve from the right ventricle to the main pulmonary (lung) artery (pulmonary valve; condition known as “pulmonic stenosis”); heartworm disease; blood clots to the lungs (known as “pulmonary embolism”); heart tumor

Nervous System and Blood Vessel Responses

- “Vasovagal syncope”—emotional stress and excitement may cause heightened sympathetic nervous system stimulation, leading to transient rapid heart rate (tachycardia) and high blood pressure (known as “hypertension”), which is followed by a compensatory response of the vagus nerve, leading to excessive enlargement or dilation of blood vessels, without a compensatory rise in heart rate and blood flow; a slow heart rate (bradycardia) often occurs
- “Situational syncope” refers to fainting (syncope) associated with coughing, defecation, urination, and swallowing
- “Carotid sinus hyperactivity” may cause low blood pressure (known as “hypotension”) and slow heart rate (bradycardia)—often the cause of fainting (syncope) when one pulls on a dog’s collar; the carotid sinus is located in the carotid artery—it acts in the regulation of blood pressure

Miscellaneous Causes

- Drugs that affect blood pressure and regulation of the autonomic nervous system; the autonomic nervous system is involved in the control of muscles in the heart, blood vessels, gastrointestinal tract, and other organs; it is composed of two parts—the sympathetic and

the parasympathetic parts; the two parts cause opposing responses; for example, the sympathetic nervous system speeds up the heart and causes the blood vessels to constrict or become small while the parasympathetic nervous system slows the heart and causes the blood vessels to expand or dilate

- Low blood sugar (known as “hypoglycemia”); low levels of calcium in the blood (known as “hypocalcemia”); and low levels of sodium in the blood (known as “hyponatremia”)
- Increased levels of red-blood cells or proteins in the blood leading to sludging of the blood [known as “hyperviscosity syndromes”] and impaired blood flow to the brain (rare)

RISK FACTORS

- Heart disease
- Sick sinus syndrome (condition in which the normal “pacemaker” function of the heart is abnormal and may lead to the heart rate being alternately slow and fast)
- Drug therapy—medications to enlarge or dilate blood vessels (known as “vasodilators,” such as calcium channel blockers, angiotensin-converting enzyme [ACE] inhibitors, hydralazine, and nitrates), phenothiazine tranquilizers (such as acepromazine), medications to control irregular heart beats (known as “antiarrhythmics”), and medications to remove excess fluid from the body (known as “diuretics”)



TREATMENT

HEALTH CARE

- Avoid or discontinue medications likely to precipitate fainting (syncope); medication should be changed only as directed by your pet’s veterinarian
- Treat as outpatient, unless important heart disease is evident

ACTIVITY

- Low blood volume being pumped by the heart (known as “low cardiac output”)—minimize activity

SURGERY

- Pacemaker implantation for some cases, such as patients with sick sinus syndrome (condition in which the normal “pacemaker” function of the heart is abnormal and may lead to the heart rate being alternately slow and fast) and advanced heart blocks



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Slow Heart Rate (Bradycardia or Bradyarrhythmia)

- Correct metabolic causes
- Medications used to increase the heart rate (known as “anticholinergics”), such as atropine, propantheline bromide, hyocamine sulfate
- Medications used to mimic the effect of the sympathetic nervous system to speed up the heart rate (known as “sympathomimetics”), such as isoproterenol and medications to enlarge or dilate the bronchi and bronchioles (known as “bronchodilators”)
- Pacemaker implantation in some patients

Rapid Heart Rate (Tachycardia or Tachyarrhythmia)

- Irregular heart beat caused by problems in the atria (known as “atrial arrhythmias”)—administer digoxin, β -blocker, or diltiazem
- Irregular heart beat caused by problems in the ventricles (known as “ventricular arrhythmias”)—administer lidocaine, procainamide, mexiletine, sotalol, or β -blocker

Low Blood Volume Being Pumped by the Heart (Low Cardiac Output)

- Institute treatment to improve blood volume being pumped by the heart (cardiac output), which varies according to specific heart disease

Nervous System and Blood Vessel Response (Vasovagal)

- Theophylline or aminophylline—sometimes helpful
- β -blockers (such as atenolol, propranolol, and metoprolol) indirectly may prevent stimulation of the vagus nerve

- Medications used to increase the heart rate (anticholinergics) may blunt the response of the vagus nerve



FOLLOW-UP CARE

PATIENT MONITORING

- Serial recordings of an electrocardiogram (“ECG,” a recording of the electrical activity of the heart) or Holter monitoring (where the patient wears a “vest” in which a continuous, mobile battery-powered ECG monitor has been placed; the ECG recording is performed over several hours, giving a better overall picture of the heart rate and rhythm) to assess effectiveness of medications to control irregular heart beat (antiarrhythmic therapy)

PREVENTIONS AND AVOIDANCE

- Minimize stimuli that precipitate episodes
- Low blood volume being pumped by the heart (low cardiac output)—minimize activity
- Vasovagal response—minimize excitement and stress
- Cough—remove collar

POSSIBLE COMPLICATIONS

- Injury when “collapse” occurs
- Death

EXPECTED COURSE AND PROGNOSIS

- Most non-heart causes are not life-threatening
- Heart causes may be treated, but fainting (syncope) in patients with heart disease may suggest higher risk of death



KEY POINTS

- Temporary loss of consciousness due to decreased oxygen reaching the brain, usually associated with abnormal circulation of blood
- Most non-heart causes are not life-threatening
- Heart causes may be treated, but fainting (syncope) in patients with heart disease may suggest higher risk of death

SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

(DISORDER IN WHICH THE IMMUNE SYSTEM ATTACKS VARIOUS BODY TISSUES)



BASICS

OVERVIEW

- “Autoimmune disease” is a disease in which the immune system attacks the animal’s own tissues
- “Antibodies” are proteins that are produced by the immune system in response to specific antigens—in the case of autoimmune disease, these antigens are located on body tissues; “antigens” are substances that induce sensitivity or an immune response
- Systemic lupus erythematosus is an autoimmune disease that involves many different body systems; it is characterized by the formation of antibodies against a wide array of body antigens
- Also known as “SLE”

GENETICS

- Inherited in a colony of German shepherd dogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dog breeds that may be more likely to develop systemic lupus erythematosus (SLE) than other breeds—Shetland sheepdog, collie, German shepherd dog, Old English sheepdog, Afghan hound, beagle, Irish setter, and poodle
- Cat breeds that may be more likely to develop systemic lupus erythematosus (SLE) than other breeds—Persian, Siamese, and Himalayan

Mean Age and Range

- Mean age is 6 years, but systemic lupus erythematosus (SLE) can occur at any age

Predominant Sex

- None

SIGNS/OBSERVED CHANGES in the ANIMAL

- Onset of signs can be sudden (acute) or subtle (insidious)
- Depend on the location in the body (such as skin or joints) where the immune system is attacking the body
- Signs vary in intensity—they may increase and decrease over time (known as a “waxing and waning” course) with clinical signs often occurring sequentially
- Sluggishness (lethargy)
- Lack of appetite (known as “anorexia”)
- Shifting-leg lameness; “shifting-leg” lameness is characterized by lameness in one leg, then that leg appears to be normal and another leg is involved
- Swollen and/or painful joints—major presenting sign in most patients
- Symmetrical or localized skin lesions—redness of the skin (known as “erythema”); scaling; superficial loss of tissue on the surface of the skin, frequently with inflammation (known as “ulceration”); loss of pigment in the skin and/or hair coat (known as “depigmentation”); and/or loss of hair (known as “alopecia”)
- Fever—especially in the sudden (acute) phase of the disease
- Superficial loss of tissue on the surface (ulceration) of the areas of the body where the skin and moist tissues of the body meet (areas known as “mucocutaneous junctions”) and on the moist tissues of the mouth (known as “oral mucosa”) may develop
- Enlargement of lymph nodes (known as “lymphadenopathy”) and of the liver and spleen (known as “hepatosplenomegaly”)
- Muscle pain or wasting
- Nervous system signs

CAUSES

- Definitive causes unidentified

RISK FACTORS

- Exposure to ultraviolet light may increase the severity of the disease



TREATMENT

HEALTH CARE

- Hospitalization—may be necessary for initial management (such as in a patient with rapid breakdown of red-blood cells [known as a “hemolytic crisis”])
- Outpatient management—often possible
- Supportive care varies with body systems affected

ACTIVITY

- Enforced rest—during episodes of sudden (acute) inflammation of several joints (known as “polyarthritis”)
- Avoid sunlight if sensitization to light (known as “photosensitization”) is suspected

DIET

- Protein restriction—recommended in animals with glomerulonephritis; “glomerulonephritis” is inflammation and accompanying dysfunction of glomeruli (plural of glomerulus) of the kidney; each kidney is composed of thousands of nephrons (the functional units of the kidney, each consisting of the glomerulus [a tuft of blood capillaries—the “blood filter”] and a series of tubes and ducts, through which the filtered fluid flows, as urine is produced); inflammation most commonly is due to the presence of immune complexes in the glomerulus



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids—to control the abnormal immune response and reduce inflammation; example is prednisone
- Chemotherapeutic drugs to decrease the immune response—may be added to the treatment when prednisone fails to improve the condition after 7 to 10 days or if the patient is (or is expected to be) steroid intolerant; possible drugs include azathioprine, cyclophosphamide, or chlorambucil
- Levamisole—also may be useful in achieving remission and can be combined with lower dose of prednisone
- Cyclosporine (Neoral® formulation)—may be tried in patients that do not respond to other medications; use with caution, and withdraw if side effects occur; requires measurement of blood cyclosporine concentration at regular intervals



FOLLOW-UP CARE

PATIENT MONITORING

- Physical examination—weekly
- Blood work, including a complete blood count (CBC) and serum biochemical analysis—to monitor the side effects of chemotherapeutic drugs on Day 7, then CBC and possible liver enzymes every second week until tapering drug dosages
- Antinuclear antibody (ANA)—the antinuclear antibody test measures antibodies in the blood to the nuclei of cells—it is used in the diagnosis of systemic lupus erythematosus (SLE); often remains elevated during remission, but may fall as patient improves clinically

PREVENTIONS AND AVOIDANCE

- Do not breed affected animals

POSSIBLE COMPLICATIONS

- Kidney failure and nephrotic syndrome (a medical condition in which the animal has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin) secondary to glomerulonephritis (inflammation and accompanying dysfunction of glomeruli [plural of glomerulus] of the kidney)

- Pneumonia or the presence of pus-forming bacteria and their poisons in the blood or tissues (known as “sepsis”) secondary to decrease of the immune response (known as “immunosuppression”)

EXPECTED COURSE AND PROGNOSIS

- Prognosis is guarded
- The presence of low red-blood cell count due to the breakdown of red-blood cells (known as “hemolytic anemia”) and glomerulonephritis (inflammation and accompanying dysfunction of glomeruli [plural of glomerulus] of the kidney) and the development of bacterial infection warrant a poor prognosis



KEY POINTS

- Systemic lupus erythematosus (SLE) is a progressive and unpredictable disease
- The patient needs long-term treatment to decrease the immune response (known as “immunosuppressive therapy”)
- Immunosuppressive therapy has potentially serious side effects; discuss the side effects with your pet’s veterinarian
- Systemic lupus erythematosus (SLE) has been shown to have a genetic basis in a colony of German shepherd dogs; potentially it could be inherited in other animals

PRIMARY IMMUNE-MEDIATED LOW PLATELET OR THROMBOCYTE COUNT (THROMBOCYTOPENIA)



BASICS

OVERVIEW

- “Platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- The range for normal platelet counts in dogs and cats generally is 200,000/ μ l to 500,000/ μ l of blood (although reference ranges can vary in different medical laboratories)
- “Thrombocytopenia” is a low platelet count, indicating a small number of platelets are present in the circulating blood
- “Primary immune-mediated thrombocytopenia” is destruction of platelets by the immune system, with no identifiable cause
- Secondary immune-mediated low platelet count (thrombocytopenia) involves antibodies (proteins produced by the immune system in response to a specific antigen) that are dependent on or are initiated by non-platelet antigens (substances that induce sensitivity or immune response)

GENETICS

- Genetic susceptibility is suggested by high number of cases seen in several breeds

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Common in dogs
- Rare in cats

Breed Predilection

- Cocker spaniels, poodles, and Old English sheepdogs, but any breed can be affected

Mean Age and Range

- Mostly middle-aged dogs
- Reported age range is 4 months to 17 years

Predominant Sex

- Twice as common in females (spayed or intact) than in males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Some cases have no clinical signs and the low platelet counts have been found on pre-surgical blood work
- Lack of appetite (known as “anorexia”), sluggishness (lethargy), weakness; may or may not have signs of bleeding
- Nose bleed (known as “epistaxis”); bloody stools (known as “hematochezia”); bleeding from the moist tissues of the body (known as “mucosal hemorrhages”)
- Small, pinpoint areas of bleeding (known as “petechia”); bruises or purplish patches, due to bleeding (known as “ecchymoses”) in the moist tissues of the body or in the skin
- Bleeding in or around the eyes, blindness
- Black, tarry stools due to the presence of digested blood (known as “melena”); vomiting blood (known as “hematemesis”)
- Pale gums and moist tissues of the body (known as “mucous membrane pallor”)
- Nervous system signs
- Fever, enlarged liver and spleen (known as “hepatosplenomegaly”), and enlarged lymph nodes (known as “lymphadenomegaly”) are unusual

CAUSES

- Unknown

RISK FACTORS

- May be preceded by a stressful event



TREATMENT

HEALTH CARE

- Uncomplicated cases with mild signs, low bleeding risk and good owner compliance may be treated as outpatients
- Patients with platelet counts less than 20,000/ μ l have very high risk of bleeding and warrant strict refinement (cage rest)
- Intensive nursing care may be needed based on patient signs, low blood volume (known as “hypovolemia”), central nervous system signs
- Low blood volume (hypovolemia) or low red-blood cell count (known as “anemia”) can be managed by administration of crystalloids (fluids that contain electrolytes [chemical compounds, such as sodium, potassium, chloride] necessary for the body to function, crystalloids generally are similar to the fluid content [plasma] of the blood and move easily between the blood and body tissues, example is lactated Ringer’s solution); colloids (fluids that contain larger molecules that stay within the circulating blood to help maintain circulating blood volume, examples are dextran and hetastarch); or packed red-blood cell or whole-blood transfusions
- Platelet transfusions have limited use; mainly used to stabilize bleeding in the central nervous system (brain and spinal cord) or to support unavoidable surgery

ACTIVITY

- Patients with platelet counts less than 20,000/ μ l have very high risk of bleeding and warrant strict refinement (cage rest)
- Strict rest is important to minimize bleeding

SURGERY

- Increased risk of bleeding in dogs with platelet counts of less than 30,000/ μ l
- Surgical removal of the spleen (known as “splenectomy”) is an option for cases that do not respond to medical treatment; discuss the risks and benefits of surgery with your pet’s veterinarian



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids—prednisone or prednisolone or dexamethasone
- Vincristine is a chemotherapy drug; it often is added only for cases that do not respond to steroid treatment
- In one study, starting vincristine with prednisone at presentation decreased the time for platelets to exceed 40,000/ μ l and hospitalization time by 2 days each without observed side effects
- Adding other medications to decrease the immune response (known as “immunosuppressive medications,” such as cyclophosphamide, azathioprine, danazol, cyclosporine) with steroids for initial treatment in dogs has not been demonstrated to be more effective than steroids alone or in combination with vincristine
- Sucralfate (medication that forms a protective barrier over the ulcer) and antacids can be administered, if stomach ulcers are suspected
- Cats usually respond well to steroids (prednisone or prednisolone) at doses that decrease the immune response (immunosuppressive doses)



FOLLOW-UP CARE

PATIENT MONITORING

- Perform platelet counts daily to every few days, until platelet numbers exceed 50,000/ μ l, then weekly until platelet numbers normalize
- Platelet counts should be performed weekly or every 2 weeks during the period of gradually tapering medications

PREVENTIONS AND AVOIDANCE

- Use modified-live (MLV) vaccines judiciously—role of MLV vaccines in recurrence of immune-mediated thrombocytopenia is

uncertain

- Minimize stress that may initiate recurrence

POSSIBLE COMPLICATIONS

- Death from hemorrhagic shock (where the blood volume has decreased through bleeding to the point that circulation or blood flow is unable to sustain the body) or central nervous system bleeding
- Ulcers of the stomach and/or intestines
- Opportunistic infections (infection caused by a organism that usually does not cause disease, but is able to cause disease because the animal's body and/or immune system has been weakened by some other disease process)
- Approximately 20% of dogs with primary immune-mediated low platelet count (thrombocytopenia) also have immune-mediated hemolytic anemia; "immune-mediated hemolytic anemia" is the destruction of red-blood cells by the immune system, which allows the release of hemoglobin (the compound in the red-blood cells that carries oxygen to the tissues of the body)

EXPECTED COURSE AND PROGNOSIS

- For primary immune-mediated thrombocytopenia, platelet counts usually will increase to 50,000/ μ l to 100,000/ μ l by 7 to 10 days of starting steroid treatment
- Some dogs never achieve normal platelet counts and require long-term (chronic) maintenance treatment
- Failure to respond to treatment should prompt reconsideration of the diagnosis
- Approximately 50% of dogs experience only one episode of immune-mediated low platelet counts (thrombocytopenia)
- The mortality rate for dogs is approximately 30%



KEY POINTS

- "Platelets" and "thrombocytes" are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to "plug" tears in the blood vessels and to stop bleeding
- "Thrombocytopenia" is a low platelet count, indicating a small number of platelets are present in the circulating blood
- "Primary immune-mediated thrombocytopenia" is destruction of platelets by the immune system, with no identifiable cause
- Strict rest is important to minimize bleeding
- Patients that develop severe bleeding, seizures, or changes in mental status should be hospitalized
- Unnecessary medications and nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided

BLOOD CLOTS IN THE LUNGS (PULMONARY THROMBOEMBOLISM)



BASICS

OVERVIEW

- “Pulmonary” refers to the lungs; “thromboembolism” is a condition in which blood flow is blocked secondary to the presence of a blood clot in the artery
- Develops when a blood clot lodges in one of the pulmonary arteries and blocks blood flow to the portion of lung served by that artery

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Disease may be more common in medium- and large-breed dogs

Mean Age and Range

- More frequently seen in middle-age to older dogs
- Bimodal age distribution (that is, having two distinct peaks of occurrence) reported in the cat, with peak occurrence in cats less than 4 years of age and in cats greater than 10 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Signs often reflect the primary, underlying disease
- Very sudden difficulty breathing (known as “peracute dyspnea”)
- Rapid breathing (known as “tachypnea”)
- Lack of appetite (known as “anorexia”)
- Collapse
- Cough or spitting up of blood derived from the lungs due to pulmonary or bronchial hemorrhage (known as “hemoptysis”)
- Weakness
- Exercise intolerance
- Inability to sleep or get comfortable
- Rapid heart rate (known as “tachycardia”)
- Weak pulses
- Pale gums or moist tissues of the body (known as “mucous membranes”)
- Bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (condition known as “cyanosis”)
- Pink color of the gums is slow to return when the gums are blanched by finger pressure (known as “poor capillary refill time”)
- Abnormal heart sounds in severely affected animals

CAUSES

- Heartworm disease
- Cancer
- Excessive levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”)
- Protein-losing enteropathy and nephropathy (conditions in which proteins are lost from the body through the intestines [enteropathy] or kidneys [nephropathy])
- Immune-mediated hemolytic anemia—accelerated destruction or removal of red-blood cells related to an immune response, in which the body produces antibodies against red-blood cells
- Inflammation of the pancreas (known as “pancreatitis”)
- Heart disease
- Bone (orthopedic) trauma or surgery
- Generalized bacterial infection (known as “sepsis”)
- Blood-clotting disorder (known as “disseminated intravascular coagulopathy” or “DIC”)

- Liver disease

RISK FACTORS

- Blood-clotting disorders (known as “coagulopathies”)
- Estrogen administration
- Airplane travel



TREATMENT

HEALTH CARE

- Always treat underlying disease
- Always treat patients suspected of having blood clots in the lungs (pulmonary thromboembolism) as inpatients until low levels of oxygen in the blood (known as “hypoxemia”) are resolved
- Cautious administration of intravenous fluids to avoid fluid overload and possible development of right-sided congestive heart failure; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Administer oxygen, if difficulty breathing (dyspnea) exists and/or oxygen levels in the blood are low; response to oxygen therapy is variable

ACTIVITY

- Restrict to prevent worsening of low levels of oxygen in the blood (hypoxemia) or fainting (known as “syncope”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Heparin is an anticoagulant (a medication to prevent blood from clotting) that may help to prevent further blood clots from developing; it will not cause existing clots to break up
- Medications to break up existing clots (known as “thrombolytic drugs,” such as urokinase, streptokinase or tissue plasminogen activator) may be useful in some cases; these drugs are expensive and carry a higher risk of bleeding complications
- Warfarin (an anticoagulant)—usually indicated for long-term treatment; dosage adjustments are necessary to keep blood clotting at a specific level to prevent development of further clots, while avoiding bleeding complications
- The “low molecular weight heparins” are associated with fewer bleeding complications, require less intensive monitoring, and may be more suitable for long-term management; however, the expense of these drugs may be a limiting factor in their use



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor serial arterial blood gases (measurements of oxygen and carbon dioxide levels in arterial blood) and/or pulse oximetry (a means of measuring oxygen levels in blood)—may help determine improvement in breathing function
- Check clotting status (using a blood test, known as “prothrombin time” or “PT”) every 3 days initially for adjusting warfarin dosage; check weekly after an effective dosage is achieved (typically no sooner than 2 weeks)

PREVENTIONS AND AVOIDANCE

- Activity or physical therapy may improve blood flow and prevent development of blood clots in immobile patients with severe generalized (systemic) disease
- Aspirin may have some preventive role, but is inadequate as treatment; aspirin should only be administered under the direction of your pet’s veterinarian
- Clopidogrel is an alternative anti-platelet drug to keep platelets from clumping together and thus decreases likelihood of clot

development; it may have some role in prevention; dose not established in the dog; “platelets” are normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body—platelets act to “plug” tears in the blood vessels and to stop bleeding; if they accumulate in a blood vessel, they may lead to a blood clot (known as “thrombosis”)

- Heparin may be administered to animals that are likely to develop blood clots in the lungs (pulmonary thromboembolism) to prevent blood clots
- Alternatively, dalteparin may be used to prevent development of blood clots

POSSIBLE COMPLICATIONS

- Bleeding complications may arise in patients treated with medications to prevent blood from clotting (anticoagulant drugs)
- Death

EXPECTED COURSE AND PROGNOSIS

- Generally guarded to poor; depends on resolution of the underlying cause
- Prognosis is somewhat better for patients with blood clots in the lungs (pulmonary thromboembolism) due to trauma or generalized bacterial infection (sepsis)



KEY POINTS

- Blood clots in the lungs (pulmonary thromboembolism) are often fatal; further episodes are likely unless an underlying cause is identified and corrected; sudden death is not unusual
- Treatment with traditional medications to prevent blood clotting (anticoagulant drugs) can lead to bleeding complications, necessitating frequent reevaluation of clotting times
- Administration of medications to prevent blood clotting (anticoagulant drugs) may be required for several months, even after resolution of the causative, underlying disease

TICK AND TICK CONTROL



BASICS

OVERVIEW

- Dogs and cats may be parasitized by ticks; ticks found on dogs and cats are in the families “*Ixodidae*” and “*Argasidae*”
- Ectoparasites are parasites that live on the surface of the host animal, such as on the skin
- Ticks are ectoparasites that feed only on the blood of their hosts; they are arthropods, closely related to scorpions, spiders, and mites
- Ticks can carry many disease-causing organisms that they transmit to their host animal—the disease-causing organisms include protozoa, bacteria, rickettsiae, and viruses; diseases caused by organisms carried by ticks are called “tick-borne diseases”
- Ticks may cause other health problems, including allergic reactions (hypersensitivity), paralysis, and blood-loss anemia

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats
- Cats are thought to be quite efficient at removing ticks, but tick attachment and subsequent health problems due to infection with disease-causing organisms carried by the tick and transmitted to the cat (tick-borne diseases) are diagnosed routinely

SIGNS/OBSERVED CHANGES in the ANIMAL

- Attached ticks or tick feeding cavities may be seen on the skin
- Associated tick-borne diseases—numerous signs, vary with the organ system(s) affected
- Irritation caused by ticks and subsequent self-trauma (as from scratching or biting at the site of tick attachment)

CAUSES

- Ticks—attracted to hosts by warmth, presence of carbon dioxide, physical contact, and host-associated odors

RISK FACTORS

- All dogs and cats exposed to ticks carrying disease-causing organisms are at risk for tick attachment and transmission of infection
- Domestic animals—can be in close contact with ticks, owing to movement of ticks into suburban environments and expansion of suburban environment into surrounding forests, prairies, and coastline areas



TREATMENT

HEALTH CARE

- Outpatient, after removal of ticks
- Removal—do as soon as possible to limit time available for transmission of the disease-causing organism or nervous-system poison (known as a “neurotoxin”) from the tick to the dog or cat; grasp ticks close to the skin with fine-pointed tweezers and gently pull free; wash feeding cavity (area of tick attachment) with soap and water; generally sufficient to prevent local inflammation or secondary infection



FOLLOW-UP CARE

PREVENTIONS AND AVOIDANCE

- Avoid environments that harbor ticks; may be difficult except for pets kept strictly indoors (and in some cases, ticks will be found indoors)
- Tick control does not always equal control of tick-borne diseases; often the goal is the perceived absence of ticks on the host animal (clinical repellence)
- Pets—owners report complete tick control, even though some period of attachment and tick feeding has occurred or live ticks may

spend some time crawling on the animal after the ticks have been exposed to lethal levels of an agent or chemical designed to kill ticks (known as an “acaricide”); immature ticks of some species that parasitize dogs and cats (*Amblyomma americanum* [the “lone star tick”], *Rhipicephalus sanguineus* [the “brown dog tick”] and *Ixodes scapularis* [the “black-legged” or “deer tick”]) may be undetected because of their tiny size

- Disease-causing organisms carried by ticks—may be transmitted very rapidly (viruses) or may require several hours (such as for *Rickettsia rickettsii* that causes Rocky Mountain spotted fever) or days (such as for *Anaplasma phagocytophilum* that requires one day for transmission or for *Borrelia burgdorferi* [cause of Lyme disease] that requires 1 to 2 days for transmission)

Insecticides and Acaricides (chemicals to kill insects and ticks)

- In the United States, the Environmental Protection Agency (EPA) licenses topical agents as effective against various species of pests
- Tick control is challenging because ticks are dispersed widely in the environment, spend a relatively short time on their hosts, possess great reproductive capacities, and have long lifespans
- Collars containing chemicals to kill ticks (known as “acaricidal collars,” such as Preventic® Tick Collar for Dogs, Virbac) and spot-on treatments (such as Frontline Topspot®, Merial and K9 Advantix®, Bayer)—have gained wide use; ease of application and owner compliance is as important as effectiveness—in many areas year-round tick control is required (NOTE: always read the entire label of any chemical designed to kill insects or ticks and use it only as instructed; *do not* use dog products on cats)
- Disease transmission interruption studies have been published for these products; at approximately 4 weeks after product application effectiveness in prevention of transmission of *B. burgdorferi* (organism that causes Lyme disease) to dogs was 75% to 87.5% for Frontline Topspot® and 100% for K9 Advantix®; Preventic® Tick Collar for Dogs was 100% effective at 7 days post-application

POSSIBLE COMPLICATIONS

- Tick-borne diseases or tick paralysis

EXPECTED COURSE AND PROGNOSIS

- Depends on which disease-causing organism has infected the dog or cat or if the nervous-system poison carried by the tick has affected the animal



KEY POINTS

- Removal of ticks—do as soon as possible to limit time available for transmission of the disease-causing organism or nervous-system poison (known as a “neurotoxin”) from the tick to the dog or cat; grasp ticks close to the skin with fine-pointed tweezers and gently pull free; wash feeding cavity (area of tick attachment) with soap and water; generally sufficient to prevent local inflammation or secondary infection
- Application of hot matches, Vaseline®, or other materials not only fails to cause tick detachment, but allows for longer periods of attachment and feeding
- Tick control is challenging because ticks are dispersed widely in the environment, spend a relatively short time on their hosts, possess great reproductive capacities, and have long lifespans

TICK PARALYSIS



BASICS

OVERVIEW

- “Lower motor neuron paralysis” is the loss of voluntary movement caused by disease of the nerves that connect the spinal cord and muscles
- “Tick paralysis” is a lower motor neuron paralysis, characterized by relaxed muscles or muscles without tone (known as “flaccid paralysis”); caused by nerve toxins found in the saliva of females of certain tick species
- Also known as “tick-bite paralysis”

GENETICS

- No genetic basis

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- United States—dogs; cats appear to be resistant
- Australia—dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Pet walked in a wooded area approximately 1 week before onset of signs
- Onset—gradual; starts with unsteadiness and weakness in the rear legs

Disease Caused by a Non-Ixodes Tick

- Once nervous system signs appear, rapidly ascending (that is, moving from rear legs to front legs and then head) lower motor neuron weakness (known as “paresis”) to paralysis
- Patient becomes recumbent in 1 to 3 days, with decreased reflexes (known as “hyporeflexia”) to lack of reflexes (known as “areflexia”) and decreased muscle tone (known as “hypotonia”) to lack of muscle tone (known as “atonia”)
- Pain sensation is preserved
- Cranial nerve dysfunction—not a prominent feature; may note facial weakness and reduced jaw tone; sometimes a change in voice (known as “dysphonia”) and difficulty swallowing (known as “dysphagia”) may be seen early in the course of disease; the “cranial nerves” are nerves that originate in the brain and go to various structures of the head (such as the eye, face, and tongue)
- Paralysis of breathing muscles (known as “respiratory paralysis”)—uncommon in cases in the United States; may occur in severely affected patients
- Urination and defecation usually are normal

Disease Caused by an Ixodes Tick

- Nervous system signs—much more severe and rapidly progressive; ascending motor weakness (that is, moving from rear legs to front legs and then head) can progress to paralysis within a few hours
- Excessive salivation, enlarged esophagus (the tube running from the throat to the stomach; condition known as “megaesophagus”), and vomiting (forceful ejection of stomach contents up through the esophagus and mouth) or regurgitation (passive, backward movement or return of food or other contents from the esophagus into the throat or mouth) are characteristic
- Dilated and poorly responsive pupils
- High blood pressure (known as “hypertension”); rapid, irregular heart beats (known as “tachyarrhythmias”)
- Fluid build-up in the lungs (known as “pulmonary edema”)
- Paralysis of breathing muscles—much more common than non-*Ixodes* related tick paralysis; dogs and cats progress to difficulty breathing (known as “dyspnea”); bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”); and respiratory paralysis within 1 to 2 days, if not treated

CAUSES

United States

- *Dermacentor variabilis*—common wood tick
- *Dermacentor andersoni*—Rocky Mountain wood tick
- *Amblyomma americanum*—lone star tick
- *Amblyomma maculatum*—Gulf Coast tick

Australia

- *Ixodes holocyclus*—Australia paralysis tick; secretes a far more potent nerve toxin than that of the North American species

RISK FACTORS

- Environments that harbor ticks
- United States—*Dermacentor variabilis*: wide distribution over the eastern two-thirds of the country and in California and Oregon; *Dermacentor andersoni*: from the Cascades to the Rocky Mountains; *Amblyomma americanum*: from Texas and Missouri to the Atlantic Coast; *Amblyomma maculatum*: the Atlantic and Gulf of Mexico seaboards
- Australia—limited to the coastal areas of the east; especially associated with areas of bush and scrub



TREATMENT

HEALTH CARE

- Inpatient—any nervous system dysfunction suggesting tick paralysis; hospitalize until either a tick is found and removed or appropriate treatment to kill a hidden tick is performed
- Inpatient supportive care—essential until patient begins to show signs of recovery
- Oxygen cage—for cases with decreased ability to breath (known as “hypoventilation”) and low levels of oxygen in the body (known as “hypoxia”)
- Artificial ventilation—for cases with breathing failure or respiratory paralysis
- Intravenous fluid therapy—generally not required, unless recovery is prolonged

ACTIVITY

- Keep patient in a quiet environment
- *Ixodes* tick paralysis—keep patient in a cool, air-conditioned area; toxin is temperature sensitive; avoid activity to prevent increase in body temperature

DIET

- Withhold food and water, if patient has difficulty swallowing (dysphagia) or vomiting/regurgitation



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- United States—if the tick cannot be found, treat the patient with an insecticidal dip, following directions for use on the product label; often the only treatment needed
- Australia—must neutralize circulating nerve toxin, depending on severity of clinical signs; if severe, phenoxybenzamine, an α -adrenergic antagonist appears to be beneficial; acepromazine can be used as an alternative medication



FOLLOW-UP CARE

PATIENT MONITORING

- Non-*Ixodes* tick—reassess nervous system status after tick removal at least daily—should see rapid improvement in muscle strength
- *Ixodes* tick—monitor nervous system status and breathing and circulatory functions continuously and intensively even after tick removal, because of the residual effect of the nerve toxin

PREVENTIONS AND AVOIDANCE

- Vigilantly check for ticks after possible exposure (at least every 2 to 3 days); signs do not occur for 4 to 6 days after tick attachment
- Weekly insecticidal baths or the use of insecticide-impregnated collars may help prevent tick paralysis (by keeping ticks off the animal or by killing the ticks before the nerve toxin has reached a level in the animal’s body to cause signs)

- Short-term immunity develops after exposure to *Ixodes* nerve toxin

POSSIBLE COMPLICATIONS

- No long-term complications, if the patient survives the sudden (acute) effects of the nerve toxin
- Death

EXPECTED COURSE AND PROGNOSIS

- Non-*Ixodes* tick—prognosis good to excellent, if ticks are removed; recovery occurs in 1 to 3 days
- *Ixodes* tick—prognosis often guarded; recovery prolonged; death in 1 to 2 days, without treatment



KEY POINTS

- Non-*Ixodes* tick—good nursing care is essential, although the patient's recovery is rapid after removal of ticks
- *Ixodes* tick—signs often continue to worsen despite tick removal; thus more aggressive treatment to neutralize the nerve toxin must be undertaken

DISEASE CAUSED BY *TOXOPLASMA*, A TYPE OF PROTOZOA (TOXOPLASMOSIS)



BASICS

OVERVIEW

- “Toxoplasmosis” is a disease caused by *Toxoplasma gondii*
- *Toxoplasma gondii*—a protozoan parasite that infects nearly all mammals; cats are the definitive hosts (meaning that reproduction of *Toxoplasma gondii* occurs in cats, with the release of one form of the parasite [known as an “oocyst”] that sporulates and becomes infective); all other warm-blooded animals are intermediate hosts (they maintain cysts in various tissues, such as muscle; infection occurs when another animal or person eats the cyst-containing tissue, such as raw or undercooked meat)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats and dogs
- Cats are more commonly symptomatic than dogs

Mean Age and Range

- In one study, mean age was 4 years; range, 2 weeks to 16 years

Predominant Sex

- Male cats—more common

SIGNS/OBSERVED CHANGES in the ANIMAL

- Determined mainly by site and extent of organ damage
- Sudden (acute) disease—at the time of initial infection
- Long-term (chronic) disease—reactivation of infection (encysted *Toxoplasma* organisms); caused by decreased ability to produce a normal immune response (known as “immunosuppression”), which allows the cyst to rupture and for *Toxoplasma* organisms to infect new cells
- Nonspecific signs of sluggishness (lethargy), depression, and lack of appetite (known as “anorexia”)
- Weight loss
- Fever
- Discharge from the eyes, avoidance of light (known as “photophobia”), constricted or narrowed pupils (known as “miotic pupils”) in cats
- Breathing distress
- Nervous system signs—wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”); seizures; tremors; weakness (known as “paresis”) or paralysis; cranial nerve deficits; the “cranial nerves” are nerves that originate in the brain and go to various structures of the head (such as the eye, face, and tongue)
- Digestive tract signs—vomiting; diarrhea; abdominal pain; yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”)
- Stillborn kittens

Cats

- Most severe in kittens infected across the placenta; kittens may be stillborn or die before weaning
- Surviving kittens—lack of appetite (anorexia); sluggishness (lethargy); high fever unresponsive to antibiotics; inflammation of lungs leading to difficulty breathing (known as “dyspnea”) or increased noises while breathing; abnormalities of the liver, leading to yellowish discoloration to the gums and other tissues of the body (jaundice or icterus) and possible abdominal enlargement from fluid build-up (known as “ascites”); and central nervous system signs, if the infection involves the brain
- Respiratory and gastrointestinal disease following birth—most common; lack of appetite (anorexia); sluggishness (lethargy); high fever unresponsive to antibiotics; difficulty breathing (dyspnea); weight loss; yellowish discoloration to the gums and other tissues of the body (jaundice or icterus); vomiting; diarrhea; build-up of fluid in the abdomen (ascites)
- Nervous system disease following birth—seen in less than 10% of patients; blindness; stupor; incoordination; circling; contraction of the neck muscles, pulling the head to one side (known as “torticollis”); unequal size of the pupils (known as “anisocoria”); seizures
- Signs involving the eyes—common; inflammation of the iris (pigmented part of the eye) and other areas in the front part of the eye (known as “uveitis”); blood in the anterior chamber of the eye (the front part of the eye, between the cornea and the iris; accumulation of blood known as “hyphema”); dilated pupils (known as “mydriasis”); inflammation of the iris (known as “iritis”); separation of the

back part of the eye (retina) from the underlying, vascular part of the eyeball (known as the “choroid;” condition known as “retinal detachment”); aggregates of inflammatory cells adhering to various areas of the inner lining of the cornea (known as “corneal endothelium;” condition known as “keratic precipitates”)—the cornea is the clear outer layer of the front of the eye

- Rapid course of disease—suddenly (acutely) affected patient with central nervous system and/or respiratory involvement
- Slow course of disease—patients with reactivation of long-term (chronic) infection

Dogs

- Young dogs—usually generalized infection; fever; weight loss; lack of appetite (anorexia); inflammation of the tonsils (known as “tonsillitis”); difficulty breathing (dyspnea); diarrhea; vomiting
- Old dogs—tend to have localized infections; mainly associated with the muscles and nervous system
- Nervous system disease—signs are quite variable; usually reflect widespread (diffuse) nervous system inflammation; seizures; tremors; wobbly, incoordinated or “drunken” appearing gait or movement (ataxia); weakness (paresis); paralysis; muscle weakness
- Signs involving the eyes—rare; similar to those found in cats
- Heart involvement—occurs; usually not clinically apparent

CAUSES

- *Toxoplasma gondii*—a protozoan parasite

RISK FACTORS

- Inability to develop a normal immune response (immunosuppression)—may increase likelihood of infection or reactivation of infection—feline leukemia virus (FeLV), feline immunodeficiency virus (FIV), feline infectious peritonitis (FIP), disease caused by a blood parasite (hemobartonellosis), canine distemper virus, and administration of steroids or chemotherapy drugs, or following a kidney transplant



TREATMENT

HEALTH CARE

- Usually outpatient
- Inpatient—severe disease; patient cannot maintain adequate nutrition or hydration
- Dehydration—intravenous fluids

ACTIVITY

- Confine—patients with nervous system signs

DIET

- Maintain adequate nutrition and hydration



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotics—clindamycin for at least 2 weeks after clinical signs resolve; sulfadiazine in combination with pyrimethamine for 2 weeks (can cause depression, low red-blood cell count [known as “anemia”], low white-blood cell count [known as “leukopenia”], and low platelet count [known as “thrombocytopenia”], especially in cats); “platelets” and “thrombocytes” are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding
- 1% prednisone drops applied to the eye directly (known as “topical treatment”)—for 2 weeks to treat inflammation of the iris (pigmented part of the eye) and other areas in the front part of the eye (uveitis)
- Folinic acid or brewer’s yeast—used to correct the decreased ability of the bone marrow to produce blood cells (known as “bone-marrow suppression”) caused by sulfadiazine/pyrimethamine treatment



FOLLOW-UP CARE

PATIENT MONITORING

Treatment with the Antibiotic, Clindamycin

- Examine 2 days after initiation of treatment—clinical signs (such as fever, lack of appetite [anorexia], inflammation of the iris [pigmented part of the eye] and other areas in the front part of the eye [uveitis]) should begin to resolve; uveitis should resolve completely within 1 week
- Examine 2 weeks after initiation of treatment—assess muscles and nervous system; signs should resolve at least partially (some signs may be permanent)
- Examine 2 weeks after owner-reported resolution of signs—assess discontinuing treatment; some signs may be permanent

PREVENTIONS AND AVOIDANCE

Cats

- Diet—prevent ingestion of raw meat, bones, viscera, or unpasteurized milk (especially goat's milk); or mechanical vectors (such as flies, cockroaches); feed only well-cooked meat
- Behavior—prevent free roaming to hunt prey (such as birds or rodents) or to enter buildings where food-producing animals are housed

POSSIBLE COMPLICATIONS

- Stillborn kittens or death of kittens
- Residual nervous system signs
- Death

EXPECTED COURSE AND PROGNOSIS

- Prognosis—guarded; varied response to drug treatment
- Sudden (acute) disease—prompt and aggressive therapy often successful
- Residual deficits (especially nervous system signs) cannot be predicted until after a course of therapy; some signs may be permanent
- Eye disease—usually responds to treatment
- Severe muscular or nervous system disease—usually results in long-term (chronic) debility



KEY POINTS

- Cats—prognosis guarded in patients needing therapy; response to therapy is inconsistent
- Newborn animals and animals that cannot develop a normal immune response (severely immunocompromised animals)—prognosis is worse
- Considerable zoonotic potential; “zoonotic potential” refers to diseases that can be passed from animals to people
- Infected cats may shed oocysts in their bowel movement
- Avoid contact with oocysts or tissue cysts—do not feed raw meat; wash hands and surfaces (cutting boards) after preparing raw meat; boil drinking water, if source is unreliable; keep sandboxes covered to prevent cats from defecating in them; wear gloves when gardening; wash hands and vegetables before eating to avoid contact with oocyst soil contamination; empty cat litter boxes daily (oocysts need at least 24 hours to become infective); disinfect litter boxes with boiling water; control stray cat population to avoid oocyst contamination of environment
- Pregnant women—talk to your veterinarian and physician about what you can do to protect yourself from infection with *Toxoplasma*; understanding how the disease is spread and what you should do will protect your baby; you should avoid all contact with a cat that is excreting oocysts in feces; avoid contact with soil and cat litter; do not handle or eat raw meat (to kill organism, cook to 66° C [150° F]); it is not necessary to give up your cat—practice good hygiene and take proper precautions for you and your baby

TRACHEAL COLLAPSE

(ABNORMALITY OF THE WINDPIPE)



BASICS

OVERVIEW

- The windpipe or trachea is the large airway that carries air from the nose and throat to the airways (bronchi) that go to the lungs
- “Tracheal collapse” is a reduction in the diameter of the lumen of the windpipe (trachea) during breathing; it is considered to be a “dynamic” process as the lumen’s diameter changes with the movements of breathing (inspiration and expiration)
- May involve the windpipe (trachea) in the neck (known as the “cervical trachea”), the windpipe (trachea) within the chest (known as the “intrathoracic trachea”), or both segments
- Compression of the windpipe (trachea) or bronchi as a result of enlarged lymph nodes or the presence of tumors are not considered part of this condition

GENETICS

- Unknown

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Primarily dogs, rarely cats

Breed Predilection

- Miniature poodles, Yorkshire terriers, Chihuahuas, Pomeranians, and other small- and toy-breed dogs
- Occasionally seen in young, large-breed dogs

Mean Age and Range

- Middle-aged to elderly—onset of signs at 4 to 14 years of age
- Severely affected animals may be less than 1 year of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Usually worsened by excitement, heat, humidity, exercise, or obesity
- Dry, honking cough
- May have long-term (chronic) intermittent coughing or difficulty breathing
- Retching (attempting to vomit)—often observed; occurs from an attempt to clear respiratory secretions from the voice box (larynx)
- Rapid breathing (known as “tachypnea”), exercise intolerance, and/or severe breathing difficulty (known as “respiratory distress”)—common
- Severe breathing difficulty (respiratory distress)—seen during inspiration (breathing in) with collapse of the windpipe in the neck (cervical tracheal collapse); seen during expiration (breathing out) with collapse of the windpipe within the chest (intrathoracic tracheal collapse)
- Bluish discoloration of the skin and moist tissues (mucous membranes) of the body caused by inadequate oxygen levels in the red-blood cells (known as “cyanosis”) or fainting (known as “syncope”)—may see in severely affected individuals
- Increased tracheal sensitivity
- Whistling sounds (wheezing) or musical sounds over the narrowed area of the windpipe may be heard while listening with a stethoscope (known as “auscultation”)
- A “snap” sound may be heard (when listening with a stethoscope) at the end of expiration, when large segments of the windpipe (trachea) collapses within the chest (intrathoracic tracheal collapse) during forceful expiration
- Abnormal breath sounds on listening to the lungs with a stethoscope (auscultation)—increased intensity or breath sounds over the bronchi; short, rough snapping sounds (known as “crackles”); and squeaking or whistling sounds (known as “wheezes”)—indicate coexistent small airway disease
- Heart murmurs (mitral valve insufficiency murmurs)—often are found in small-breed dogs with tracheal collapse
- Normal to low heart rate—common in dogs with tracheal collapse, unless severe breathing difficulty (respiratory distress) occurs
- Loud second heart sound detected when listening to the heart with a stethoscope (auscultation)—suggests increased blood pressure within the lungs (known as “pulmonary hypertension”)
- Enlarged liver (known as “hepatomegaly”)—cause unknown

CAUSES

- Defects in the development of cartilage in the windpipe (trachea)
- Long-term (chronic) small-airway disease

RISK FACTORS

- Obesity
- Infection or inflammation of the lungs
- Upper airway blockage or obstruction



TREATMENT

HEALTH CARE

- Outpatient—stable patients
- Inpatient—oxygen therapy and heavy sedation for severe breathing difficulty (respiratory distress) or for severely anxious patients

ACTIVITY

- Severely limited, until patient is stable
- During management of disease—gentle exercise recommended to encourage weight loss

DIET

- Most affected dogs improve after losing weight
- Institute weight-loss program with a high-fiber reducing diet
- Feed 60% of total daily requirement of calories; use a slow weight-loss program

SURGERY

- Surgery—may benefit some patients, primarily those with collapse of the windpipe (trachea) in the neck (cervical tracheal collapse)
- Signs due to upper airway obstructive disorder (such as paralysis of the voice box or larynx [known as “laryngeal paralysis”], turning inside-out of a portion of the voice box or larynx [known as “everted laryngeal sacculs”])—may improve after corrective surgery
- Placement of stents to keep the lumen of the windpipe open, in selected patients (primarily with collapse of the windpipe [trachea] in the neck [cervical tracheal collapse]) by a skilled surgeon—shown to improve quality of life and reduce clinical signs when adequate stabilization of the airway can be achieved and when long-term (chronic) lung changes do not limit resolution of disease
- Consider likelihood of complications after surgery (such as persistent cough, severe breathing difficulty [respiratory distress], or paralysis of the voice box (larynx; laryngeal paralysis)); some patients may require a permanent surgical opening into the windpipe or trachea (known as a “permanent tracheostomy”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Sedation and cough suppression—butorphanol; addition of a tranquilizer (acepromazine) may enhance sedative effects and further reduce the cough reflex; narcotic cough suppressants (butorphanol or hydrocodone) effective for long-term (chronic) treatment
- Drugs to dilate the bronchi and bronchioles (known as “bronchodilators”); dilation of small airways and lowering pressure gradients with lower airway disease—sustained-release theophylline or terbutaline; bronchodilators have no effect on the diameter of the windpipe (trachea)
- Reduction of inflammation of the windpipe (trachea)—prednisone; consider inhaled steroids given via face mask and spacer chamber
- Robitussin® DM—may provide relief to animal, reduce the severity of the cough, but is not a cure (known as “palliation”)



FOLLOW-UP CARE

PATIENT MONITORING

- Body weight
- Exercise tolerance
- Pattern of breathing
- Incidence of cough

PREVENTIONS AND AVOIDANCE

- Avoid obesity in breeds commonly afflicted with tracheal collapse
- Avoid heat and humidity
- Use a harness rather than a collar (a collar puts pressure on the windpipe, and may aggravate the problem)

POSSIBLE COMPLICATIONS

- Severe breathing difficulties that do not respond to medical treatment (known as “intractable respiratory distress”) leading to respiratory failure or euthanasia

EXPECTED COURSE AND PROGNOSIS

- Combinations of medications, along with weight control, may reduce clinical signs
- Surgery—may benefit some patients, primarily those with collapse of the windpipe (trachea) in the neck (cervical tracheal collapse)
- Patient will cough throughout life
- Prognosis—based on evidence and degree of airway blockage



KEY POINTS

- “Tracheal collapse” is a reduction in the diameter of the lumen of the windpipe (trachea) during breathing; it is considered to be a “dynamic” process as the lumen’s diameter changes with the movements of breathing (inspiration and expiration)
- Obesity, over excitement, and humid conditions may precipitate a breathing crisis
- Use a harness instead of a collar
- Combinations of medications, along with weight control, may reduce clinical signs

INFECTIOUS TRACHEOBRONCHITIS (KENNEL COUGH)



BASICS

OVERVIEW

- Any contagious respiratory disease of dogs that is manifested by coughing, and seemingly not caused by canine distemper virus or canine influenza virus

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilection

- None

Mean Age and Range

- Most severe in puppies 6 weeks to 6 months old
- May develop in dogs of all ages and often with pre-existing subclinical airway disease (such as abnormal development of the respiratory tract or long-term inflammation of the bronchi (known as “chronic bronchitis”))

SIGNS/OBSERVED CHANGES in the ANIMAL

- Related to the degree of respiratory tract damage and age of the affected dog
- May be nonexistent, mild, or severe with pneumonia
- Most viral, bacterial, and *Mycoplasma* agents spread rapidly from seemingly healthy dogs to other dogs in the same environment; signs usually begin about 4 days after exposure to the infecting agent(s)
- Uncomplicated—cough in an otherwise healthy animal is characteristic; may be dry and hacking, soft and dry, moist and hacking, or sudden and sharp, followed by gagging or spitting up of mucus; excitement, exercise, changes in temperature or humidity of the inspired air, and gentle pressure (such as from collar) on the windpipe (trachea) induce a sudden onset of coughing
- Uncomplicated—cough readily induced with pressure on the windpipe (trachea) during physical examination; lung sounds often normal; otherwise appears healthy
- Severe—marked loss of appetite; cough (when noted) is moist and productive; may see sluggishness (lethargy), difficulty breathing (known as “dyspnea”), and exercise intolerance
- Severe—may have constant, low-grade, or fluctuating fever (39.4 to 40.0° C; 103 to 104° F); may have increased intensity of normal lung sounds; short, rough lung sounds (known as “crackles”) heard with a stethoscope; or (less frequently) whistling or squeaking sounds (known as “wheezes”)

CAUSES

- Viral—canine adenovirus-2; canine parainfluenza virus; canine adenovirus-1; canine reovirus type 1, 2, or 3; canine herpesvirus
- Canine adenovirus-2 and canine parainfluenza virus may damage the lining cells of the respiratory tract to such an extent that invasion by various bacteria and *Mycoplasma* cause severe airway disease
- Bacterial—*Bordetella bronchiseptica*, (with no other respiratory disease-causing agents) produces clinical signs indistinguishable from those of other bacterial causes; *Pseudomonas*, *Escherichia coli*, *Klebsiella*, *Pasteurella*, *Streptococcus*, *Mycoplasma*, and other species equally likely

RISK FACTORS

- Dogs housed with multiple other dogs, such as pet shops, humane society shelters, research facilities, and boarding and training kennels
- Pre-existing subclinical airway disease, such as abnormal development of the respiratory tract or long-term inflammation of the bronchi (known as “chronic bronchitis”)



TREATMENT

HEALTH CARE

- Outpatient—strongly recommended for uncomplicated disease

- Inpatient—strongly recommended for complicated disease and/or pneumonia
- Fluid administration—indicated for complicated disease and/or pneumonia

ACTIVITY

- Enforce rest—for at least 14-to-21 days with uncomplicated disease; for at least the duration of X-ray evidence of pneumonia

DIET

- Good-quality canned or dry commercial food



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Antibiotic therapy—amoxicillin/clavulanic acid or doxycycline—initial treatment of uncomplicated disease
- Antibiotic therapy—gentamicin, amikacin, or enrofloxacin—and a first-generation cephalosporin (such as cefazolin) usually effective for severe pneumonia; continue antibiotic therapy for at least 10 days beyond resolution of pneumonia as seen on X-rays
- *Bordetella bronchiseptica*—some antibiotics may not reach adequate levels in the lower respiratory tract to be effective, so administration of these antibiotics by mouth or injection may have limited effectiveness; treating with a fine medicated spray (known as “nebulization”) containing kanamycin, gentamicin, or polymyxin B may reduce bacterial numbers, when administered daily for 3 to 5 days
- Cough suppressants (such as butorphanol or hydrocodone)—effective in decreasing the dry, nonproductive cough, when infection has been controlled
- Drugs to increase the openings in the bronchi and bronchioles (known as “bronchodilators,” such as extended-release theophylline)—may be used to control narrowing of the bronchi and bronchioles due to contraction of smooth muscles in the walls of these airways (known as “bronchospasm”); bronchospasm is detected clinically by whistling or squeaking sounds (wheezes)



FOLLOW-UP CARE

PATIENT MONITORING

- Uncomplicated disease—should respond to treatment in 10 to 14 days; if patient continues to cough 14 days or more after adequate treatment, dog should be evaluated again by your pet’s veterinarian
- Severe disease—repeat chest X-rays until at least 14 days beyond resolution of all clinical signs

PREVENTIONS AND AVOIDANCE

- Shedding of the causative virus and/or bacteria of infectious tracheobronchitis (kennel cough) in respiratory secretions of dogs undoubtedly accounts for the persistence of this problem in kennels, animal shelters, boarding facilities, and veterinary hospitals; thorough cleaning and disinfecting of kennels is necessary to control spread of disease-causing organisms

Viral and Bacterial Vaccines

- Available to control disease caused by the principal infectious agents involved
- *Bordetella bronchiseptica* and canine parainfluenza virus vaccine—may vaccinate puppies using a vaccine applied into the nose (intranasal vaccine) as early as 2 to 4 weeks of age, without interference from maternal antibody; follow-up vaccinations should be administered as directed by your pet’s veterinarian; may vaccinate mature dogs with a one-dose intranasal vaccination (at the same time as their puppies or when they receive other vaccinations, as directed by your pet’s veterinarian)
- Inactivated injectable *Bordetella bronchiseptica* vaccine—administered as two doses, 2 to 4 weeks apart; initial vaccination of puppies is recommended at or about 6 to 8 weeks of age; administer second vaccine at 4 months of age

EXPECTED COURSE AND PROGNOSIS

- Natural course of uncomplicated disease, if untreated—10 to 14 days; simple restriction of exercise and prevention of excitement shortens the course
- Typical course of severe disease—2 to 6 weeks; may be fatal in patients that develop severe pneumonia, affecting multiple lung lobes



KEY POINTS

- Isolate patient from other animals; infected dogs can transmit the disease-causing virus and/or bacteria before onset of clinical signs and afterward until immunity develops
- Patients with uncomplicated disease should respond to treatment in 10 to 14 days
- Once infection spreads in a kennel, it can be controlled by removing all dogs from the premises for 1 to 2 weeks and disinfecting with commonly used chemicals, such as sodium hypochlorite (bleach; 1:30 dilution), chlorhexidine, or benzalkonium (NOTE: never mix disinfectants; follow directions for use carefully)

TRANSITIONAL CELL CARCINOMA OF THE URINARY TRACT (KIDNEY, URETERS, BLADDER, URETHRA)



BASICS

OVERVIEW

- The urinary tract consists of the kidneys, the ureters (the tubes running from the kidneys to the bladder), the urinary bladder (that collects urine and stores it until the animal urinates), and the urethra (the tube from the bladder to the outside, through which urine flows out of the body)
- Transitional cell carcinoma is a cancer arising from the transitional epithelium of the kidney, ureters, urinary bladder, urethra, prostate, or vagina; the transitional epithelium is a specialized type of lining in the urinary tract that contracts or stretches in response to the size of the bladder and other organs

GENETICS

- Possible genetic basis in Scottish terriers

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Dogs—Scottish terriers, West Highland white terriers, Shetland sheepdogs, American Eskimo dogs, and dachshunds; may occur in any breed
- Cats—none

Mean Age and Range

- Dogs—8 years; range, 1 to 15+ years of age
- Cats—7 years; range, 3 to 16 years of age
- Middle-aged to old, spayed, female small-breed dogs most commonly reported

Predominant Sex

- Female

SIGNS/OBSERVED CHANGES in the ANIMAL

- Similar to those of bacterial urinary tract infection; for patients showing temporary or no response to appropriate antibiotics, consider transitional cell carcinoma; may temporarily respond to antibiotic therapy
- Recurrent straining with slow, painful discharge of urine (known as “stranguria”); abnormal frequent passage of urine (known as “pollakiuria”); blood in the urine (known as “hematuria”); difficulty urinating (known as “dysuria”); inability to control urination or leaking urine (known as “urinary incontinence”); or any combination of these signs
- Physical examination findings often normal
- Mass—occasionally may be felt in the abdomen at the location of the urinary bladder
- Urethral or vaginal transitional cell carcinoma—may be able to feel mass during rectal examination
- Enlarged intrapelvic or sublumbar lymph nodes—may be able to feel during rectal examination

CAUSES

- Dogs—obesity; environmental carcinogens (substances that cause cancer); long-term (chronic) exposure to flea-control products; and long-term treatment or a large bolus dose of cyclophosphamide (a chemotherapeutic drug)
- Cats—unknown



TREATMENT

HEALTH CARE

- Outpatient—stable patients

- Seek advice from a veterinary oncologist prior to initiating treatment and consider current recommendations
- Radiation therapy: intraoperative radiation therapy is reported to result in longer survival times and better local control than chemotherapy; potential side effects: narrowing (stricture) and scarring (fibrosis) of the bladder with lack of control of urination or urine leakage (urinary incontinence)

ACTIVITY

- Normal

DIET

- Normal, unless animal also has kidney failure

SURGERY

- Surgery for transitional cell carcinoma can be challenging as the tumor easily sheds cancer cells; these cells can be spread into the abdomen during surgery
- Surgery may result in a cure, if the mass can be removed completely
- Wide surgical excision (that is, surgically removing the tumor and wide borders of apparently normal tissue) is necessary; up to 50% of the urinary bladder may be removed surgically with minimal loss of function
- Placement of a catheter from the bladder and exiting through the abdominal wall to allow urine to be removed from the body (procedure known as “tube cystostomy”)—may greatly prolong survival times by bypassing blockage of the urethra (known as “urethral obstruction”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Chemotherapy—piroxicam (Feldene®), cisplatin, carboplatin, mitoxantrone (as single agents or as combination therapy for certain drugs); piroxicam and cisplatin cannot be combined because of kidney toxicity; other agents (doxorubicin or doxorubicin/cyclophosphamide combination) may have activity
- Antibiotics—administered as necessary for secondary urinary tract infections



FOLLOW-UP CARE

PATIENT MONITORING

- X-rays using contrast media in the bladder (known as “contrast cystography”) or ultrasound examination—every 6 to 8 weeks; assess response to treatment and screen for spread of cancer into the lymph nodes (known as “lymph-node metastases”)
- Chest X-rays—every 2 to 3 months; detect spread of cancer into the lungs (known as “pulmonary metastatic disease”)

POSSIBLE COMPLICATIONS

- Blockage of the urethra (the tube from the bladder to the outside, through which urine flows out of the body) or ureters (the tubes running from the kidneys to the bladder), and kidney failure
- Spread of cancer (metastasis) to regional lymph nodes, lungs, or bone
- Recurrent urinary tract infection
- Lack of control of urination or urine leakage (urinary incontinence)
- Reduction of bone-marrow activity (known as “myelosuppression”), resulting in low number of red-blood cells, white-blood cells, and/or platelets or gastrointestinal toxicity secondary to chemotherapy
- Gastrointestinal ulceration secondary to piroxicam therapy

EXPECTED COURSE AND PROGNOSIS

- Long-term prognosis grave
- Progressive disease probable
- Median survival—no treatment, 4 to 6 months; with treatment, 6 to 12 months



KEY POINTS

- Long-term prognosis is poor, but control of signs to make the animal more comfortable (known as “palliation”) is often attainable
- The tumor usually cannot be removed surgically in dogs

TREMORS



BASICS

OVERVIEW

- Repetitive, rhythmic, oscillatory (swinging back and forth), involuntary movements of all or part of the body
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇; thoracic vertebrae are located from the area of the shoulders to the end of the ribs and are numbered as thoracic vertebrae one through thirteen or T₁-T₁₃; lumbar vertebrae start at the end of the ribs and continue to the pelvis and are numbered as lumbar vertebrae one through seven or L₁-L₇; the remaining vertebrae are the sacral and coccygeal (tail) vertebrae

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Decreased amounts or absence of myelin, the protective covering of many nerve fibers (condition known as “hypomyelination”)—chow chows, English springer spaniels, Samoyeds, Weimaraners, and Dalmatians
- Brief head tremor of unknown cause (so called “idiopathic transient head tremor”)—Doberman pinschers, English bulldogs, and Labrador retrievers

Mean Age and Range

- Age depends on cause
- Generalized tremor syndrome—usually young to middle-aged dogs
- Decreased amounts or absence of myelin, the protective covering of many nerve fibers (hypomyelination)—6 to 8 weeks of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Localized or generalized tremors
- Localized tremors—most often involve the head or the rear legs

CAUSES

Head Tremors

- Abnormality of part of the brain, the cerebellum—degenerative; congenital (present at birth); inflammatory; immune-mediated; toxic causes
- Unknown cause (so called “idiopathic disease”)—Doberman pinschers and English bulldogs are more likely to develop head tremors of unknown cause than other breeds
- Genetic
- Inflammatory—inflammation of the brain (known as “encephalitis”)
- Trauma
- Drug administration—doxorubicin (a chemotherapy drug); diphenhydramine (an antihistamine); metoclopramide (medication used to control nausea and vomiting)

Rear Leg Tremors

- May be a sign of weakness or pain in the lumbosacral area of the spine
- Metabolic—kidney failure; inadequate production of parathyroid hormone, leading to abnormalities in blood calcium and phosphorus levels (condition known as “hypoparathyroidism”); decreased levels of glucose (sugar) in the blood (known as “hypoglycemia”)
- Compressive lesions of the spine or nerve roots—narrowing of the spinal canal in the lumbosacral spine (known as “lumbosacral stenosis”); pressure to or damage of the nerves within the spinal canal in the area of the junction between the lumbar and sacral vertebrae—at this level of the spine, spinal nerves are located in the spinal canal (rather than spinal cord), these spinal nerves within the spinal canal are known as the “cauda equina” (condition known as the “cauda equina syndrome”); spinal cord tumor; bacterial or fungal infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition known as “diskospondylitis”)
- Disorder involving the nerves to the rear legs (known as a “peripheral neuropathy”); neuromuscular junction abnormality; disorder involving the muscles of the rear legs (known as a “myopathy”)
- Poor blood flow to the muscles of the rear legs—right-to-left shunting of blood flow through a birth defect, known as a “patent ductus arteriosus;” other diseases of the heart and lungs

- Unknown cause (so called “idiopathic disease”)—rear leg tremors in older dogs (so called “senile tremor”)

Generalized Tremors

- Decreased amounts or absence of myelin, the protective covering of many nerve fibers (hypomyelination)
- Poisons—organophosphates (type of insecticide); hexachlorophene (an antiseptic product); bromethalin (product used to kill rodents)
- Degenerative nervous system disease—inherited metabolic diseases in which harmful levels of materials accumulate in the body’s cells and tissues (known as “storage diseases”); a disorder characterized by progressive deterioration of nervous tissue, causing the formation of numerous tiny holes in the brain (known as “spongiform encephalopathy”)
- Generalized tremors of unknown cause (so called “idiopathic generalized tremor syndrome”)—generalized body tremors seen in young, predominantly small-breed dogs, initially described in white dogs (such as Maltese and West Highland white terriers, leading to the name, “white shaker dog syndrome”)

RISK FACTORS

- Any inflammation of the brain (encephalitis) or degenerative nervous system disease—inherited metabolic diseases in which harmful levels of materials accumulate in the body’s cells and tissues (storage disease); a disorder characterized by progressive deterioration of nervous tissue, causing the formation of numerous tiny holes in the brain (spongiform encephalopathy)
- Treatment with doxorubicin (a chemotherapy drug); diphenhydramine (an antihistamine); metoclopramide (medication used to control nausea and vomiting)



TREATMENT

HEALTH CARE

- Treat the underlying primary disease
- Outpatient, unless surgical treatment is indicated (such as lumbosacral disease that requires decompression and stabilization)
- Generalized tremor of primary brain origin—patient may lose weight; monitor weight and modify oral intake accordingly
- Drug-induced tremors—consider an alternate drug
- Suspected poisoning—remove pet from further exposure; contact your pet’s veterinarian immediately; consult with a poison control center for possible antidote

SURGERY

- Surgery may be indicated for some causes of tremor (such as diseases involving the lumbosacral spine)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Usually do not respond to muscle relaxants or drugs to control seizures (such as phenobarbital or diazepam)
- Steroids—to decrease the immune response (known as giving an “immunosuppressive dose” of steroids) to treat generalized tremor syndrome
- Antibiotics—for bacterial infection of the intervertebral disks and adjacent bone of the spine (vertebral bodies; condition is diskospondylitis); chosen on the basis of bacterial culture and sensitivity testing results of samples from the spinal lesion, blood, or urine
- Diseases of part of the brain, the cerebellum—depends on the diagnosis
- Gabapentin may be helpful in treatment of some tremors



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor the primary disease

- Steroids for generalized tremor syndrome—monitor weekly initially to assess response to treatment

PREVENTIONS AND AVOIDANCE

- Avoid excitement and exercise—may worsen many tremors

EXPECTED COURSE AND PROGNOSIS

- Most causes of tremors in adult dogs are treatable
- Degenerative nervous system diseases (such as inherited metabolic diseases in which harmful levels of materials accumulate in the body's cells and tissues [storage disease]; a disorder characterized by progressive deterioration of nervous tissue, causing the formation of numerous tiny holes in the brain [spongiform encephalopathy])—no treatment available
- Decreased amounts or absence of myelin, the protective covering of many nerve fibers (hypomyelination)—generally not treatable; some breeds (such as the chow chow) improve with maturity
- Head tremor of unknown cause (idiopathic head tremor)—no effective treatment available; benign tremor that occurs sporadically; has few health consequences



KEY POINTS

- Repetitive, rhythmic, oscillatory (swinging back and forth), involuntary movements of all or part of the body
- Localized tremors—most often involve the head or the rear legs
- Most causes of tremors in adult dogs are treatable

FUNCTIONAL URINARY RETENTION (INCOMPLETE EMPTYING OF THE BLADDER)



BASICS

OVERVIEW

- Incomplete emptying of the urinary bladder (voiding), which is not associated with physical blockage of the lower urinary tract (known as “urinary obstruction”)
- The lower urinary tract consists of the urinary bladder and the urethra (the tube from the bladder to the outside, through which urine flows out of the body)
- The detrusor muscle is the muscular layer of the wall of the urinary bladder; it contracts to empty the bladder and to cause urine to leave the body through the urethra
- “Functional” is defined as being caused by a problem with the normal action of an organ

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Predominant Sex

- More common in male than in female dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Distended urinary bladder may be felt on physical examination; after attempts by the animal to empty the bladder (void), distension may persist or an excessive amount of urine may remain in the bladder (the inappropriate residual urine volume can be measured)
- Affected animals may demonstrate ineffective, frequent, or no attempts to urinate (void)
- Urine stream may be weak, thin or diminished (attenuated), or interrupted
- Abdominal distension, abdominal pain, or signs of excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) due to inability to remove urine from the body (known as “postrenal azotemia”) may predominate in rare cases or with urinary tract rupture
- May contribute to recurrent urinary tract infection; functional urinary retention may be identified because of the recurrent infection
- Lack of control of urination or urine leakage due to overflow of urine (known as “overflow urinary incontinence”) may occur

CAUSES

Decreased Contraction (Hypocontractility) of the Urinary Bladder Detrusor Muscle (known as “Detrusor Atony”)

- Most commonly develops as a sequel to sudden (acute) or long-term (chronic) urinary bladder overdistension; many patients have a history of nervous system dysfunction or previous urinary blockage or obstruction
- Nervous system causes include lesions of the pelvic nerves, sacral spinal cord (the sacral spinal cord is located within the sacral vertebrae, which are found within the pelvis just before the vertebrae of the tail), and suprasacral spinal cord (that is, before the sacral spinal cord; for example, the lumbar spinal cord)
- Lesions of the sacral spinal cord (such as congenital malformations, cauda equina compression, lumbosacral disk disease, and vertebral fractures/dislocations) can result in a flaccid, overdistended urinary bladder with weak outlet resistance (outlet resistance is inhibition of the ability to pass urine through the urethra)
- Lesions of the suprasacral spinal cord (such as intervertebral disk protrusion, spinal fractures, and compressive tumors) can result in a distended, firm urinary bladder that is difficult to express or empty by gentle manual pressure
- Electrolyte disturbances, including excessive levels of potassium in the blood (known as “hyperkalemia”); inadequate levels of potassium in the blood (known as “hypokalemia”); excessive levels of calcium in the blood (known as “hypercalcemia”); inadequate levels of calcium in the blood (known as “hypocalcemia”); and other metabolic disturbances associated with generalized muscle weakness also can affect contraction of the urinary bladder detrusor muscle (detrusor muscle contractility)
- Decreased contraction of the detrusor muscle (detrusor atony) with urine retention is a feature of a disorder characterized by abnormal function of the autonomic nervous system (known as “dysautonomia”); dysautonomia is encountered primarily in cats in Great Britain; the disorder also has been described in dogs in certain geographic regions of the United States
- Some dogs with excessive levels of steroids produced by the adrenal glands (known as “hyperadrenocorticism” or “Cushing’s disease”) have increased urination (known as “polyuria”), urinary bladder distention and mild urine retention

Functional Urinary Obstruction (blockage or obstruction of urination related to abnormal action of the lower urinary tract)

- Occurs when excessive or inappropriate outlet resistance (inhibition of the ability to pass urine through the urethra) prevents complete

emptying of the bladder (voiding) during urinary bladder contraction

- In patients with suprasacral spinal lesions or midbrain disorders, urethral outlet resistance (inhibition of the ability to pass urine through the urethra) becomes uninhibited and remains inappropriately excessive or fails to coordinate with voiding contractions (that is, the contraction of the detrusor muscle and the relaxation of the urethra are not coordinated, known as “detrusor-urethral dyssynergia”); the condition has been associated with sacral lesions, local nervous system disease (known as “neuropathy”), and unknown causes (so called “idiopathic” disease)
- Excessive urethral resistance (inhibition of the ability to pass urine through the urethra), usually attributed to spasm of muscular components of the urethra (known as “urethrospasm”), may be seen after urethral blockage or obstruction or urethral or pelvic surgery, urethral inflammation, or prostatic disease

RISK FACTORS

- Feline lower urinary tract disease (FLUTD)
- Urethral blockage or obstruction
- Pelvic or urethral surgery
- Anticholinergic medications, such as atropine



TREATMENT

HEALTH CARE

- Usually managed as inpatients, until adequate emptying of the bladder (voiding) returns
- Address primary disorders (such as electrolyte disturbances and nervous system lesions) and correct, if possible
- Manage excess levels of urea and other nitrogenous waste products in the blood (uremia or azotemia), electrolyte imbalances, and acid–base disturbances associated with sudden (acute) urine retention
- Identify urinary tract infection, and treat appropriately
- Keep the urinary bladder small by intermittent or indwelling catheterization or frequent manual compression; intermittent or indwelling urinary catheterization may be required temporarily to ensure urine flow

SURGERY

- May consider surgical options for salvaging the opening of the urethra in some patients; surgical removal of the penis and creation of a new opening in the urethra (known as “perineal urethrostomy”) may be required in male cats with unmanageable urethral resistance (inhibition of the ability to pass urine through the urethra) in the end of the urethra



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Decreased Contraction (Hypocontractility) of the Urinary Bladder Detrusor Muscle (Detrusor Atony)

- Bethanechol—may increase contraction of the detrusor muscle in partially denervated or suddenly (acutely) overdistended urinary bladders
- Metoclopramide—may stimulate detrusor muscle contraction
- Cisapride—may promote bladder emptying

Functional Urinary Obstruction (blockage or obstruction of urination related to abnormal action of the lower urinary tract)

- Prazosin or phenoxybenzamine— α -adrenergic antagonists; reduces smooth muscle contraction in the urethra; more effective in dogs than in cats
- Diazepam—a short-acting, central skeletal muscle relaxant; relaxes striated muscle of the external urethral sphincter, the muscle that opens the urethra when the animal urinates (voids)
- Acepromazine—a phenothiazine tranquilizer; has general muscle relaxant and α -blocking effects on urethral tone; may be effective in cats with excessive urethral resistance (inhibition of the ability to pass urine through the urethra)
- Dantrolene—a muscle relaxant; appears to be effective in reducing distal urethral resistance (inhibition of the ability to pass urine through the urethra) in cats
- Baclofen—a spinal reflex inhibitor; acts as a skeletal muscle relaxant; clinical use in small animals has been limited



FOLLOW-UP CARE

PATIENT MONITORING

- As treatment progresses, assess residual urine volume by urinary bladder palpation or by periodic urinary catheterization
- In most patients, can withdraw medications slowly after primary causes are corrected and adequate emptying (voiding) function of the bladder has been sustained for several days
- Perform periodic urinalysis in patients with long-term (chronic) urine retention to detect urinary tract infection
- Complete bladder emptying (voiding) function may not return; pets should be monitored for signs of urinary blockage or obstruction or excess levels of urea and other nitrogenous waste products in the blood (uremia or azotemia)

POSSIBLE COMPLICATIONS

- Lower urinary tract infection and infection moving up the urinary tract to the kidneys (known as “ascending infection”)
- Permanent detrusor muscle injury and decreased contraction (atony); urinary bladder or urethral rupture
- Excess levels of urea and other nitrogenous waste products in the blood (uremia or azotemia) due to inability to remove urine from the body (postrenal azotemia)

EXPECTED COURSE AND PROGNOSIS

- Depend on cause and response to treatment



KEY POINTS

- Incomplete emptying of the urinary bladder (voiding), not associated with blockage of the lower urinary tract (known as “urinary obstruction”)
- Affected animals may demonstrate ineffective, frequent, or no attempts to void
- Urine stream may be weak, thin or diminished (attenuated), or interrupted
- May contribute to recurrent urinary tract infection
- Complete bladder emptying (voiding) function may not return; pets should be monitored for signs of urinary blockage or obstruction or excess levels of urea and other nitrogenous waste products in the blood (uremia or azotemia)

CALCIUM OXALATE UROLITHIASIS

(CALCIUM OXALATE STONES IN THE URINARY TRACT)



BASICS

OVERVIEW

- “Urolithiasis” is the medical term for the presence of stones (uroliths) in the urinary tract
- The most common minerals found in the stones (uroliths) are used to name the particular stone; in this type of stone, calcium and oxalate (or oxalic acid) make up the composition of the stone, and thus the name “calcium oxalate urolithiasis”
- Calcium oxalate urolithiasis is the formation of calcium oxalate stones (known as “calcium oxalate uroliths”) within the urinary tract and associated clinical conditions
- The urinary tract consists of the kidneys, the ureters (the tubes running from the kidneys to the bladder), the urinary bladder (that collects urine and stores it until the animal urinates), and the urethra (the tube from the bladder to the outside, through which urine flows out of the body)
- In dogs, calcium oxalate accounts for approximately 40% of the stones (uroliths) removed from the lower urinary tract (bladder and urethra) and 45% of those removed from the upper urinary tract (kidneys and ureters)
- In cats, calcium oxalate accounts for approximately 40% of the stones (uroliths) removed from the lower urinary tract (bladder and urethra) and 60% of those retrieved from the upper urinary tract (kidneys and ureters)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Dogs—reported in many breeds—six breeds represent 60% of cases: miniature schnauzer, Lhasa apso, Yorkshire terrier, bichon frise, shih tzu, and miniature poodle
- Cats—Himalayan, Scottish fold, Persian, ragdoll, and Burmese are at greater risk of forming calcium oxalate stones than other breeds

Mean Age and Ranges

- Dogs—8.5 years of age; 60% of cases are 6 to 11 years of age
- Cats—97% are older than 2 years of age; 53% of cases are 7 to 15 years of age

Predominant Sex

- Mostly male dogs (73%) and male cats (55%)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Some animals have no signs of disease (known as “asymptomatic”)
- Depend on location, size, and number of urinary tract stones (uroliths)
- Animals with stones located in the kidney(s) (known as “nephroliths”) are typically asymptomatic, but may have persistent blood in the urine (known as “hematuria”)
- The blockage or obstruction of a ureter (the tube running from the kidney to the bladder) is associated with changes in the kidneys, and sudden (acute) excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) and occur frequently in cats
- Typical signs of stones in the bladder (known as “urocystoliths”) or the urethra (the tube from the bladder to the outside, through which urine flows out of the body; stones known as “urethroliths”) include abnormal frequent passage of urine (known as “pollakiuria”); difficulty urinating (known as “dysuria”) and blood in the urine (hematuria)
- Stones in the kidneys and ureters (the tubes running from the kidneys to the bladder; stones known as “nephroureteroliths”) are common in cats with kidney failure
- Stones in the bladder (urocystoliths) may be detected during physical examination; failure to feel stones does not exclude them from consideration.
- Enlarged urinary bladder, if patient has complete blockage or obstruction of the urethra (the tube from the bladder to the outside, through which urine flows out of the body)—more common in cats
- Stones in the bladder (urocystoliths) with irregular contours uncommonly cause complete blockage or obstruction of the urethra (the tube from the bladder to the outside, through which urine flows out of the body)

CAUSES

- Presence of high levels of calcium in the urine (known as “hypercalciuria”); high levels of oxalic acid or oxalates in the urine (known

as “hyperoxaluria”); low levels of citrate in the urine (known as “hypocitraturia”); and defective crystal-growth inhibitors (substances that increase the solubility of calcium oxalate and decrease the likelihood of stone formation)

- Feeding diets that promote acidic urine (known as “aciduria”) in cats

RISK FACTORS

- Calcium supplements independent of meals
- Excessive dietary protein and vitamin D may increase the amount of calcium in the urine, leading to high levels of calcium in the urine (hypercalciuria)
- Additional dietary oxalate (such as from peanuts) and ascorbic acid may increase the amount of oxalic acid and oxalates in the urine, leading to hyperoxaluria
- High levels of steroids (either occurring naturally in the body, when the adrenal glands produce excess steroids, or by administration of steroid-containing medications), diets that promote formation of acidic urine (aciduria), and furosemide (a diuretic) may increase the amount of calcium in the urine, leading to high levels of calcium in the urine (hypercalciuria)
- Pyridoxine (vitamin B₆)-deficient diets (such as homemade diets) may increase the amount of oxalic acid and oxalates in the urine, leading to high levels of oxalic acid or oxalates in the urine (hyperoxaluria)
- Consumption of dry diets is associated with a higher risk for calcium oxalate stone (urolith) formation than consumption of high-moisture, canned diets



TREATMENT

HEALTH CARE

- Removal of the stones can be performed by flushing stones located in the urethra (the tube from the bladder to the outside, through which urine flows out of the body) back into the urinary bladder or by positioning the animal and using gentle compression of the bladder to allow the animal to urinate and “pass” the stones to eliminate small bladder stones on an outpatient basis; allowing the animal to urinate and “pass” the stones should not be tried if blockage or obstruction of the urethra exists
- Medical procedures in which the stone is broken up within the urinary tract using some type of energy or sound wave (procedures known as “laser lithotripsy” or “shock-wave lithotripsy”) and surgery require short periods of hospitalization

ACTIVITY

- Reduce activity following surgery, until healed

DIET

- Calcium oxalate stones (uroliths) do not dissolve with special diets
- Increased levels of calcium in the blood (known as “hypercalcemia”) in cats (without evidence of increased levels of parathyroid hormone and its effects [known as “hyperparathyroidism”] or cancer) is sometimes minimized by use of Hill’s Prescription Diet® Feline w/d®

SURGERY

- Consider surgical removal of lower urinary tract stones (uroliths) that cannot be removed by other means
- Shock-wave lithotripsy (in which the stone is broken up within the urinary tract using some type of energy or sound wave) is an alternative to surgery for removal of kidney stones (nephroliths) and ureter stones (ureteroliths) in dogs and bladder stones
- Consider surgical removal of the parathyroid glands (known as “parathyroidectomy”) for patients with primary hyperparathyroidism and increased levels of calcium in the blood (hypercalcemia)



MEDICATIONS

- No available drugs effectively dissolve calcium oxalate stones (uroliths)



FOLLOW-UP CARE

PATIENT MONITORING

- Postsurgical X-rays are essential to verify complete stone (urolith) removal
- To attempt to prevent the need for repeat surgery, evaluate abdominal X-rays every 3 to 5 months to detect stone (urolith) recurrence early (small stones may be removed by flushing stones located in the urethra [the tube from the bladder to the outside, through which urine flows out of the body] back into the urinary bladder or by positioning the animal and using gentle compression of the bladder to allow the animal to urinate and “pass” the stones)

PREVENTIONS AND AVOIDANCE

- If the patient has high levels of calcium in the blood (hypercalcemia), correct underlying cause
- Consider Hill’s Prescription Diet® Feline w/d® for cats with increased levels of calcium in their urine for unknown cause (condition known as “idiopathic hypercalciuria”); administer potassium citrate to minimize acidic urine (aciduria)
- If the patient has normal levels of calcium in the blood (known as “normocalcemia”), consider a diet with reduced oxalate and protein that does not promote formation of acidic urine (such as Hill’s Prescription Diet® Canine u/d®, Hill’s Prescription Diet® Feline x/d®); ideally, the diet should contain additional water (canned diets) and citrate, and have adequate quantities of phosphorus and magnesium; avoid supplementation with vitamins C and D
- Reevaluate patient 2 to 4 weeks after initiation of diet therapy to verify appropriate reduction in specific gravity of the urine (indicating a more dilute or less concentrated urine, which makes it more difficult for crystals to form), appropriate urine pH of 6.5 or higher (indicating that the urine is not in the acidic range), and decrease in crystals in the urine (known as “crystalluria”)
- To promote more dilute or less concentrated urine, consider feeding canned formulations of food or add water to all types of food
- If urine is acidic, consider adding potassium citrate; adjust dosage to achieve a urine pH between 6.5 and 7.5
- Vitamin B₆ may help minimize oxalate excretion into the urine, especially for animals fed homemade or pyridoxine (vitamin B₆)-deficient diets

POSSIBLE COMPLICATIONS

- Stones in the bladder (urocystoliths) can pass into and block the urethra (the tube from the bladder to the outside, through which urine flows out of the body) in male dogs and cats, especially if the patient is having difficulty urinating (dysuria)
- Dogs that do not consume their daily requirement of the stone (urolith) prevention diet can develop various degrees of protein-calorie malnutrition
- Diet-associated increased levels of lipids (a group of compounds that contain fats or oils, condition known as “hyperlipidemia”) develops in some patients; miniature schnauzers with inherited hyperlipidemia are more likely to develop inflammation of the pancreas (known as “pancreatitis”) when consuming the stone-prevention diet; Hill’s Prescription Diet ® Canine w/d® can be used as an alternative diet (it should be supplemented with potassium citrate, as needed, to maintain a urine pH between 6.5 and 7.5)

EXPECTED COURSE AND PROGNOSIS

- Approximately 50% of dogs with normal calcium levels in their blood (normocalcemia) reform calcium oxalate stones (uroliths) in 2 years; patients with increased levels of calcium in their blood (hypercalcemia) typically reform calcium oxalate stones at a faster rate
- Treatment to minimize recurrence of calcium oxalate stones (uroliths) is helpful
- At least 10% of cats reform calcium oxalate stones (uroliths) in 2 years



KEY POINTS

- Stone (urolith) removal does not alter the factors responsible for their formation; eliminating risk factors is necessary to minimize recurrence
- Approximately 50% of dogs with normal calcium levels in their blood (normocalcemia) reform calcium oxalate stones (uroliths) in 2 years; patients with increased levels of calcium in their blood (hypercalcemia) typically reform calcium oxalate stones at a faster rate
- Treatment to minimize recurrence of calcium oxalate stones (uroliths) is helpful
- At least 10% of cats reform calcium oxalate stones (uroliths) in 2 years

STRUVITE UROLITHIASIS IN CATS

(STRUVITE STONES IN THE URINARY TRACT OF CATS)



BASICS

OVERVIEW

- “Urolithiasis” is the medical term for the presence of stones (uroliths) in the urinary tract
- The most common minerals found in the stones (uroliths) are used to name the particular stone; in this type of stone, struvite makes up the composition of the stone, and thus the name “struvite urolithiasis;” struvite is magnesium ammonium phosphate
- The urinary tract consists of the kidneys, the ureters (the tubes running from the kidneys to the bladder), the urinary bladder (that collects urine and stores it until the animal urinates), and the urethra (the tube from the bladder to the outside, through which urine flows out of the body)
- Struvite stones (uroliths) and accumulations of struvite and inflammatory materials in a matrix (known as “struvite urethral plugs”) are different in physical characteristics and causes; thus, these terms should not be used as synonyms—struvite stones (uroliths) are crystalline concretions, composed primarily of magnesium ammonium phosphate and small quantities of matrix while struvite urethral plugs commonly are composed of large quantities of matrix mixed with crystals (especially, magnesium ammonium phosphate); some urethral plugs are composed primarily of organic matrix, sloughed tissue, blood, and/or inflammatory cells

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Mean Age and Range

- Mean age at time of diagnosis is approximately 7 years (range, less than 1 year to 22 years of age)
- Struvite stones (uroliths) that are free of the presence of microorganisms, such as bacteria (that is, “sterile struvite uroliths”), do not affect immature cats; infection-induced struvite stones may occur in immature cats

Predominant Sex

- Struvite stones (uroliths) are more common in females (55%) than in males (45%)
- Accumulations of struvite and inflammatory materials in a matrix (struvite urethral plugs) primarily affect males

SIGNS/OBSERVED CHANGES in the ANIMAL

- Some affected cats have no signs of disease (known as “asymptomatic”)
- Depend on location, size, number and cause of the stones (uroliths)
- Typical signs of stones in the bladder (known as “urocystoliths”) include abnormal frequent passage of urine (known as “pollakiuria”); difficulty urinating (known as “dysuria”) and blood in the urine (known as “hematuria”)
- Typical signs of stones in the urethra (the tube from the bladder to the outside, through which urine flows out of the body; stones known as “urethroliths”) include abnormal frequent passage of urine (known as “pollakiuria”); difficulty urinating (known as “dysuria”), and sometimes small, smooth stones (uroliths) are passed when the animal urinates (voids); signs (such as lack of appetite [anorexia] and vomiting) of excess levels of urea and other nitrogenous waste products in the blood are found in some cats with blockage or obstruction of urine flow out of the body (condition known as “postrenal uremia”)
- Signs of kidney insufficiency or failure (such as increased urination [known as “polyuria”] and increased thirst [known as “polydipsia”]) are found in some cats with stones in the kidneys (stones known as “nephroliths”)
- Signs typical of inability to urinate because of blockage or obstruction of the urethra (the tube from the bladder to the outside, through which urine flows out of the body), such as difficulty urinating (dysuria), large painful urinary bladder, and signs of postrenal uremia are found in cats with accumulations of struvite and inflammatory materials in a matrix (struvite urethral plugs)
- A thickened, firm, contracted bladder wall is detected on physical examination in some cats with stones in the bladder (urocystoliths)
- Accumulations of struvite and inflammatory materials in a matrix (struvite urethral plugs) or struvite stones in the urethra (urethroliths) may be detected during physical examination of the penis and penile urethra (the part of the urethra that is enclosed by the penis)

RISK FACTORS

- For formation of sterile struvite stones (uroliths)—include the mineral composition, energy content, and moisture content of the diet being fed; compounds in the diet that make the urine more alkaline (that is, have a higher pH); quantity of diet consumed; free-choice versus meal-feeding schedules; formation of concentrated urine; and retention of urine
- Probable for formation of infection-induced struvite stones (uroliths)—include urinary tract infection with bacteria that produce

urease, an enzyme that breaks down urea to carbon dioxide and ammonia (urea is the final compound in the breakdown of protein in the body); abnormalities in local host defenses that allow bacterial urinary tract infections; and the quantity of urea (the substrate of urease) excreted in urine

- The normal small diameter of the end of the urethra in male cats makes them susceptible to blockage with plugs and stones (urethroliths)



TREATMENT

HEALTH CARE

- Removal of the stones can be performed by flushing stones located in the urethra (the tube from the bladder to the outside, through which urine flows out of the body) back into the urinary bladder, flushing the urethra to remove accumulations of struvite and inflammatory materials in a matrix (struvite urethral plugs), or by positioning the cat and using gentle compression of the bladder to allow the cat to urinate and “pass” the stones to eliminate bladder and urethral stones, and/or surgery require short periods of hospitalization
- Dissolving the struvite stones (uroliths) medically is an outpatient strategy
- Struvite stones in the ureters (ureteroliths) or urethra (urethroliths) cannot be dissolved

ACTIVITY

- If dietary management is used, monitor outdoor activity in order to limit access to other foods and treats

DIET

- Sterile and infection-induced struvite stones in the bladder (urocystoliths) and in the kidneys (nephroliths) may be dissolved by feeding a diet designed to eliminate stones (Hill’s Prescription Diet® Feline s/d®)
- Continue diet therapy for 1 month after X-ray evidence showing that the stone (urolith) has dissolved
- Struvite crystals in the urine (crystalluria) may be minimized by feeding magnesium-restricted urine-acidifying diets
- If dietary management is used, limit access to other foods and treats
- Canned (moist) foods help to reduce urine concentration of stone-forming compounds and promote increased frequency of normal urination

SURGERY

- Struvite stones in the ureters (ureteroliths) cannot be dissolved; consider surgery for persistent ureteroliths associated with clinical signs
- Struvite stones in the urethra (urethroliths) cannot be dissolved medically; attempt removal of the stones by flushing stones located in the urethra (the tube from the bladder to the outside, through which urine flows out of the body) back into the urinary bladder or by positioning the cat and using gentle compression of the bladder to allow the cat to urinate and “pass” the stones or urethral plugs, or flushing the urethra to remove accumulations of struvite and inflammatory materials in a matrix (struvite urethral plugs)
- Immovable stones in the urethra (urethroliths), recurrent accumulations of struvite and inflammatory materials in a matrix (struvite urethral plugs), or narrowing (known as “strictures”) of the end of the urethra (the tube from the bladder to the outside, through which urine flows out of the body) may require surgical removal of the penis with creation of a new opening into the urethra (surgical procedure known as “perineal urethrostomy”)
- Medical procedure in which the stone is broken up within the urinary tract using light energy (known as “laser lithotripsy”) may be used for struvite stones in the bladder (urocystoliths) and/or the urethra (urethroliths)
- Struvite stones in the kidneys (nephroliths) causing blockage or obstruction of urine flow, or associated with nonfunctioning kidneys, cannot be dissolved medically; consider surgical correction if stones (uroliths) are blocking urine outflow, and/or if correctable abnormalities increasing the likelihood of recurrent urinary tract infection are identified by X-rays or other means
- Struvite stones (uroliths) and accumulations of struvite and inflammatory materials in a matrix (struvite urethral plugs) should be localized, before considering surgical correction
- X-rays should be obtained immediately following surgery to verify that all stones (uroliths) were removed



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Dissolving infection-induced struvite stones in the bladder (urocystoliths) or in the kidney (nephroliths) requires administration of appropriate antibiotics, chosen on the basis of bacterial culture and susceptibility tests

- Give antibiotics at therapeutic dosages, until the urinary tract infection is eradicated and no X-ray evidence of bladder stones exists
- Difficulty urinating (dysuria) may be minimized by treatment of bacterial urinary tract infection with antibiotics, and by administration of an anticholinergic drug (such as propantheline bromide) to relax the bladder



FOLLOW-UP CARE

PATIENT MONITORING

- Check rate of stone (urolith) dissolution at monthly intervals by urinalysis, urine culture, X-rays, or ultrasound
- Monitor patients, in which the urine has been acidified, for calcium oxalate crystals in the urine (crystalluria); change management protocol if persistent calcium oxalate crystalluria develops

PREVENTIONS AND AVOIDANCE

- Recurrent struvite stones (uroliths) that are free of the presence of microorganisms, such as bacteria (that is, sterile struvite uroliths) may be prevented by using acidifying, magnesium-restricted diets or urine acidifiers—do not administer urine acidifier medications with acidifying diets
- In patients at risk for both struvite and calcium oxalate crystals in the urine, focus on preventing calcium oxalate stones (uroliths); struvite uroliths may be dissolved medically; recurrent calcium oxalate uroliths cannot be dissolved
- Infection-induced struvite stones in the urinary tract (urolithiasis) can be prevented by eradicating and controlling urinary tract infections; use of magnesium-restricted, acidifying diets is another method of prevention, but often is not required

POSSIBLE COMPLICATIONS

- Struvite stones in the bladder (urocystoliths) may pass into and block the urethra (the tube from the bladder to the outside, through which urine flows out of the body) of male cats, especially if the patient persistently has difficulty urinating (dysuria); urethral blockage or obstruction may be managed by flushing stones located in the urethra back into the urinary bladder
- A urinary catheter in the urethra (known as an “indwelling transurethral catheter”) increases the risk for introduction of bacteria and resulting bacterial urinary tract infection and/or narrowing of the urethra (urethral stricture)

EXPECTED COURSE AND PROGNOSIS

- Dissolving struvite stones that are free of the presence of microorganisms, such as bacteria (that is, sterile struvite uroliths) in the bladder takes time; the mean time reported was 1 month (range, 2 weeks to 5 months)
- The mean time for dissolution of infection-induced struvite stones in the bladder (urocystoliths) was 10 weeks (range, 9 to 12 weeks)



KEY POINTS

- If dietary management is used, limit access to other foods and treats
- Short-term (weeks to months) treatment with a diet designed to eliminate stones (Hill’s Prescription Diet® Feline s/d®), with or without antibiotics (as needed), is effective in dissolving struvite stones in the kidney (nephroliths) and bladder (urocystoliths); avoid feeding these diets to immature cats
- Comply with dosage schedule for antibiotic therapy, if the cat has infection-induced struvite stones in the bladder (urocystoliths)

STRUVITE UROLITHIASIS IN THE DOG

(STRUVITE STONES IN THE URINARY TRACT OF DOGS)



BASICS

OVERVIEW

- “Urolithiasis” is the medical term for the presence of stones (uroliths) in the urinary tract
- The most common minerals found in the stones (uroliths) are used to name the particular stone; in this type of stone, struvite makes up the composition of the stone, and thus the name “struvite urolithiasis;” struvite is magnesium ammonium phosphate
- The urinary tract consists of the kidneys, the ureters (the tubes running from the kidneys to the bladder), the urinary bladder (that collects urine and stores it until the animal urinates), and the urethra (the tube from the bladder to the outside, through which urine flows out of the body)
- Struvite urolithiasis is the formation of crystalline stones (uroliths) composed of magnesium ammonium phosphate, or struvite, in the urinary tract

GENETICS

- The high incidence of struvite stones (uroliths) in some breeds of dogs (such as the miniature schnauzer) suggests a familial tendency; it is hypothesized that susceptible miniature schnauzers inherit some abnormality of local host defenses of the urinary tract that increases their likelihood to develop urinary tract infection (UTI)
- Sterile struvite uroliths were found in a family of English cocker spaniels

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predilection

- Miniature schnauzer, shih tzu, bichon frise, miniature poodle, cocker spaniel, and Lhasa apso
- Any breed may be affected

Mean Age and Range

- Mean age, 6 years (range, less than 1 year to greater than 19 years of age)
- Most stones (uroliths) in immature dogs are infection-induced struvite

Predominant Sex

- More common in females (85%) than males (15%), which may be related to the greater likelihood of females developing bacterial urinary tract infections (UTIs)

SIGNS/OBSERVED CHANGES in the ANIMAL

- Some affected dogs have no signs of disease (known as “asymptomatic”)
- Signs depend on location, size, and number of stones (uroliths)
- Typical signs of stones in the bladder (known as “urocystoliths”) include abnormal frequent passage of urine (known as “pollakiuria”); difficulty urinating (known as “dysuria”) and blood in the urine (known as “hematuria”); sometimes small, smooth stones (uroliths) are passed when the animal urinates (voids)
- Typical signs of stones in the urethra (the tube from the bladder to the outside, through which urine flows out of the body; stones known as “urethroliths”) include abnormal frequent passage of urine (known as “pollakiuria”); difficulty urinating (known as “dysuria”), and sometimes small, smooth stones (uroliths) are passed when the animal urinates (voids)
- Struvite stones of the kidney (nephroliths) may be associated with signs of kidney insufficiency or failure (such as increased urination [known as “polyuria”] and increased thirst [known as “polydipsia”])
- Obstruction to urine outflow with bacterial urinary tract infection may result in inflammation/infection of the kidney (known as “pyelonephritis”) and generalized disease caused by the spread of bacteria in the blood (known as “septicemia” or “blood poisoning”); signs might include increased urination (polyuria), increased thirst (polydipsia), abdominal or lumbar pain, and fever
- Struvite stones may be felt in the urinary bladder and/or urethra during physical examination
- Blockage or obstruction of the urethra may cause enlargement of the urinary bladder
- Obstruction of a ureter (the tube running from the kidney to the bladder) may cause enlargement of the associated kidney

CAUSES

- Urinary tract disorders that increase the likelihood of infections with bacteria that produce urease, an enzyme that breaks down urea to carbon dioxide and ammonia, disease-causing fungus; or *Ureaplasma* (a type of bacteria that breaks down urea to ammonia) in patients whose urine contains a large quantity of urea (urea is the final compound in the breakdown of protein in the body)
- Specific causes of sterile struvite stones (uroliths) are unknown; sterile struvite stones are ones that are free of the presence of microorganisms, such as bacteria

RISK FACTORS

- High concentrations of steroids (either from excess production by the adrenal glands or from use of steroid-containing medications) increase the likelihood of bacterial urinary tract infection
- Abnormal retention of urine
- Alkaline urine decreases the solubility of struvite and increases the likelihood of struvite stone formation



TREATMENT

HEALTH CARE

- Removal of the stones can be performed by flushing stones located in the urethra (the tube from the bladder to the outside, through which urine flows out of the body) back into the urinary bladder to re-establish opening of the urethra or by positioning the dog and using gentle compression of the bladder to allow the dog to urinate and “pass” the stones to eliminate bladder and urethral stones; medical procedure in which the stone is broken up within the urinary tract using some type of energy (procedure known as “shock-wave lithotripsy”) and/or surgery require short periods of hospitalization
- Medical dissolution of struvite stones (uroliths) is an outpatient strategy
- Struvite stones in the ureters (ureteroliths) or urethra (urethroliths) cannot be dissolved

ACTIVITY

- If dietary management is used, monitor outdoor activity in order to limit access to other foods and treats

DIET

- Sterile and infection-induced struvite stones in the bladder (urocystoliths) and in the kidneys (nephroliths) may be dissolved by feeding a diet designed to eliminate stones (Hill’s Prescription Diet® Canine s/d®)
- Continue diet therapy for 1 month after X-ray evidence showing that the stone (urolith) has dissolved
- If dietary management is used, limit access to other foods and treats
- Avoid use of the protein-restricted diet in patients with protein-calorie malnutrition; the diet to eliminate stones is designed for short-term (weeks to months) dissolution therapy, rather than long-term (months to years) stone prevention—if diet is used to eliminate stones, monitor the patient for evidence of protein malnutrition; avoid prolonged feeding of the diets intended to eliminate stones in immature dogs

SURGERY

- Struvite stones in the ureters (ureteroliths) cannot be dissolved; consider surgery or medical procedure in which the stone is broken up within the urinary tract using some type of energy (shock-wave lithotripsy) for persistent ureteroliths associated with clinical signs
- Struvite stones in the urethra (urethroliths) cannot be dissolved medically; attempt removal of the stones by flushing stones located in the urethra (the tube from the bladder to the outside, through which urine flows out of the body) back into the urinary bladder or by positioning the dog and using gentle compression of the bladder to allow the dog to urinate and “pass” the stones; consider medical procedure in which the stone is broken up within the urinary tract using some type of energy (shock-wave lithotripsy)
- Immovable stones in the urethra (urethroliths) may require surgery, such as a surgical incision into the urethra to reach and remove the stones (known as “urethrotomy”) or a surgical procedure in which a new permanent opening is made into the urethra to allow passage of urine out of the body (procedure known as “urethrostomy”)
- Struvite stones in the kidney (nephroliths) causing blockage or obstruction of urine flow or associated with nonfunctioning kidneys cannot be dissolved medically; consider surgical correction if stones (uroliths) are blocking urine outflow, and/or if correctable abnormalities increasing the likelihood of recurrent urinary tract infection are identified by X-rays or other means



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Dissolving infection-induced struvite stones in the bladder (urocystoliths) or in the kidney (nephroliths) requires administration of appropriate antibiotics, chosen on the basis of bacterial culture and susceptibility tests
- Give antibiotics at therapeutic dosages, until the urinary tract infection is eradicated and no X-ray evidence of bladder stones exists
- Patients with infection-induced struvite stones in the bladder (urocystoliths), associated with persistent bacterial infection with urease-producing bacteria, that are not responsive to dietary and antibiotic treatment to dissolve the stones may be given AHA (acetoxyhydroxamic acid; Lithostat™, Mission Pharmacal)—AHA is a urease inhibitor that blocks hydrolysis of urea to ammonia
- Difficulty urinating (dysuria) may be minimized by treatment of bacterial urinary tract infection with antibiotics, and by administration of an anticholinergic drug (such as propantheline bromide) to relax the bladder



FOLLOW-UP CARE

PATIENT MONITORING

- Check rate of stone (urolith) dissolution at monthly intervals by urinalysis, urine culture, X-rays, or ultrasound
- Monitor patients, in which the urine has been acidified, for calcium oxalate crystals in the urine (crystalluria); change management protocol if persistent calcium oxalate crystalluria develops

PREVENTIONS AND AVOIDANCE

- Eradicating and controlling infections by urease-producing bacteria may prevent infection-induced struvite stones
- Recurrent struvite stones (uroliths) that are free of the presence of microorganisms, such as bacteria (that is, sterile struvite uroliths) may be prevented by using acidifying, magnesium-restricted diets (Hill's Prescription Diet® Canine s/d®) or urine acidifiers
- In patients at risk for both struvite and calcium oxalate crystals in the urine, focus on preventing calcium oxalate stones (uroliths); struvite uroliths may be dissolved medically; recurrent calcium oxalate uroliths cannot be dissolved

POSSIBLE COMPLICATIONS

- Benefits and risks are associated with feeding diets designed to eliminate struvite stones; not all patients qualify for dietary medical management, including those with (1) abnormal fluid accumulation, (2) primary kidney failure with excess levels of urea and other nitrogenous waste products in the blood (known as "uremia" or "azotemia"), and (3) increased likelihood to develop inflammation of the pancreas (known as "pancreatitis"), especially miniature schnauzers with increased levels of lipids (a group of compounds that contain fats or oils, condition known as "hyperlipidemia")
- Struvite stones in the bladder (urocystoliths) may pass into and block the urethra (the tube from the bladder to the outside, through which urine flows out of the body) of male dog, especially if the patient persistently has difficulty urinating (dysuria); urethral blockage or obstruction may be managed by flushing stones located in the urethra back into the urinary bladder or by a medical procedure in which the stone is broken up within the urinary tract using some type of energy (shock-wave lithotripsy)
- Dogs that do not consume their daily requirement of the diet intended to eliminate struvite stones (uroliths) may develop varying degrees of protein-calorie malnutrition; this complication can be prevented by proper calculation of the daily dietary requirement and adjustment in the quantity of diet fed on the basis of serial physical examination
- Diet-associated increased urination (polyuria) will result in voiding increased urine volume; the increased volume may be associated with varying degrees of inability to control urination or urine leakage (known as "urinary incontinence") in spayed female dogs that have a tendency for estrogen-responsive incontinence

EXPECTED COURSE AND PROGNOSIS

- Dissolving struvite stones takes time; the mean time for dissolving infection-induced stones in the bladder (urocystoliths) was approximately 3 months (range, 2 weeks to 7 months); the mean time for dissolution of infection-induced struvite stones in the kidney (nephroliths) was 6 months (range, 2 to 10 months)
- The mean time for dissolving sterile struvite stones in the bladder was 6 weeks (range, 4 to 12 weeks)
- Following dietary recommendations is suggested by finding a reduced concentration of urea in blood testing and a low urine specific gravity (indicating dilute urine) on urinalysis
- If uroliths increase in size during dietary management or do not begin to decrease in size after approximately 4 to 8 weeks of appropriate medical management, alternative methods should be considered
- Difficulty in inducing complete dissolution of struvite stones after creating urine under-saturated with struvite should prompt consideration that (1) the wrong mineral component was identified, (2) the center (nucleus) of the stone (urolith) has a different mineral composition than other portions of the urolith, and/or (3) the owner is not complying with medical recommendations



KEY POINTS

- If dietary management is used, limit access to other foods and treats
- Short-term treatment with a diet designed to eliminate stones (Hill's Prescription Diet® Canine s/d®) and administration of antibiotics has been effective in dissolving struvite stones (uroliths)
- Comply with dosage schedule for antibiotic therapy

URATE UROLITHIASIS

(URATE STONES IN THE URINARY TRACT)



BASICS

OVERVIEW

- Urolithiasis” is the medical term for the presence of stones (uroliths) in the urinary tract
- The most common minerals found in the stones (uroliths) are used to name the particular stone; in this type of stone, uric acid or urate makes up the composition of the stone, and thus the name “urate urolithiasis”
- The urinary tract consists of the kidneys, the ureters (the tubes running from the kidneys to the bladder), the urinary bladder (that collects urine and stores it until the animal urinates), and the urethra (the tube from the bladder to the outside, through which urine flows out of the body)
- Urate stones (uroliths) are composed of uric acid, sodium urate, or ammonium urate

GENETICS

- Dalmatians have a breed susceptibility to forming urate stones (uroliths); the genetics of this condition are unknown

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- Dalmatian, English bulldog, and breeds at risk for portosystemic shunt, such as the Yorkshire terrier (“portosystemic shunt” is a condition in which abnormal blood vessels allow blood to flow between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver)

Mean Age and Range

- Mean age in patients without portosystemic shunt is 3.5 years (range, 0.5 to greater than 10 years of age)
- Mean age in patients with portosystemic shunt (condition in which abnormal blood vessels allow blood to flow between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver) is less than 1 year (range, 0.1 to greater than 10 years of age)

Predominant Sex

- More common in male dogs, in cases without a portosystemic shunt
- No sex predilection in dogs with portosystemic shunt (condition in which abnormal blood vessels allow blood to flow between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver) or in cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Some animals have no signs of disease (known as “asymptomatic”)
- Depend on location, size, and number of urinary tract stones (uroliths)
- Blood in the urine (known as “hematuria”)
- Difficulty urinating (known as “dysuria”)
- Possible signs of a nervous system disorder caused by accumulation of ammonia in the system due to inability of the liver to rid the body of ammonia (known as “hepatic encephalopathy”) in patients with portosystemic shunt (condition in which abnormal blood vessels allow blood to flow between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver); signs include sluggishness (lethargy), lack of appetite (anorexia), disorientation, blindness, seizures, coma
- Blockage or obstruction of the urethra may cause enlargement of the urinary bladder; if the blockage is complete, animal may have signs (such as lack of appetite [anorexia] and vomiting) due to excess levels of urea and other nitrogenous waste products in the blood due to the inability of the animal to urinate (condition known as “postrenal uremia”)

CAUSES

- Breed susceptibility (Dalmatian) to form urate stones (uroliths)
- Animals with portosystemic shunt (condition in which abnormal blood vessels allow blood to flow between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver) may develop urate stones (uroliths)

RISK FACTORS

- High purine (a nitrogen-containing compound) intake in the diet, especially eating glandular meat
- Persistent acidic urine (low urine pH; known as “aciduria”) in a susceptible animal



TREATMENT

HEALTH CARE

- Blockage of the urethra (the tube from the bladder to the outside, through which urine flows out of the body) or ureter (the tube running from the kidney to the bladder) may require inpatient treatment
- Urate stones (uroliths) can be dissolved on outpatient basis
- Fluid therapy to correct dehydration

ACTIVITY

- Usually not restricted, except after surgery

DIET

- For dissolution and prevention of urate stones, a low-purine, urine-alkalinizing diet (that is, a diet which makes the urine more alkaline or raises the urine pH)

SURGERY

- Surgery to remove stones from the bladder (known as “cystotomy”), urethra (known as “urethrotomy”), or kidney (known as “nephrotomy”)
- Surgery to tie off abnormal blood vessels that allow blood to flow between the portal vein (vein that normally carries blood from the digestive organs to the liver) and the body circulation without first going through the liver (known as “portosystemic shunt ligation”)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Allopurinol, a xanthine-oxidase inhibitor, for dissolving urate stones



FOLLOW-UP CARE

PATIENT MONITORING

- Monitor with urinalysis and X-ray studies or ultrasound every 1 to 2 months; if no recurrence in 6 months, evaluate every 2 to 4 months

PREVENTIONS AND AVOIDANCE

- Low-purine, urine-alkalinizing diet (that is, a diet which makes the urine more alkaline or raises the urine pH)

POSSIBLE COMPLICATIONS

- Blockage or obstruction of the urethra
- Urate stones (uroliths) likely to recur, if no preventive measures are taken

EXPECTED COURSE AND PROGNOSIS

- Dissolving the stones with medical treatment (allopurinol and diet) takes an average of 4 weeks
- Dissolving the stones with medical treatment (allopurinol and diet) usually is not successful in cases with portosystemic shunts (condition in which abnormal blood vessels allow blood to flow between the portal vein [vein that normally carries blood from the

digestive organs to the liver] and the body circulation without first going through the liver)



KEY POINTS

- Recurrence of urate stones (uroliths) is possible, especially if no preventive measures are taken
- Dalmatians have a breed susceptibility to forming urate stones (uroliths)
- Animals with portosystemic shunts (condition in which abnormal blood vessels allow blood to flow between the portal vein [vein that normally carries blood from the digestive organs to the liver] and the body circulation without first going through the liver) may develop urate stones (uroliths)

ANTERIOR UVEITIS IN CATS

(INFLAMMATION OF THE FRONT PART OF THE EYE, INCLUDING THE IRIS)



BASICS

OVERVIEW

- Inflammation of the front part of the eye, including the iris (known as “anterior uveitis”); the iris is the colored or pigmented part of the eye—it can be brown, blue, green, or a mixture of colors
- May be associated with coexistent inflammation of the back part of the eye, including the retina; the retina contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision
- May involve only one eye (known as “unilateral anterior uveitis”) or both eyes (known as “bilateral anterior uveitis”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Mean Age and Range

- Mean age—7 to 9 years
- Any age may be affected

Predominant Sex

- Males and neutered males are affected more commonly than females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Cloudy eye—due to fluid build-up in the clear part of the eye (known as “corneal edema”); cloudiness of aqueous humor (the “aqueous humor” is the transparent liquid that fills the front part of the eyeball) due to increased protein content and suspended cellular debris (condition known as “aqueous flare”); accumulation of white-blood cells in the anterior chamber of the eye (condition known as “hypopyon”)
- Painful or uncomfortable eye—signs include squinting or spasmodic blinking (known as “blepharospasm”); avoidance of light (known as “photophobia”); or rubbing the eye
- Red eye
- Vision loss—variable
- Discharge from the eye; usually excessive tearing, may have mucus and/or pus in the discharge
- Keratic precipitates—aggregates of inflammatory cells adhering to various areas of the inner lining of the cornea (known as “corneal endothelium”); the cornea is the clear outer layer of the front of the eye
- Development of blood vessels in the clear part of the eye (known as “corneal vascularization”)
- Small or constricted pupil, frequently resistant to medical treatment to dilate the pupil
- Swelling of the iris
- Decreased pressure within the eye (known as “intraocular pressure” [IOP]) is consistent with anterior uveitis, but is not seen in all cases
- Scar tissue between the iris and the lens of the eye (known as “posterior synechia”); the lens is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye (retina)
- Accumulations of white-blood cells (hypopyon), red-blood cells (known as “hyphema”), or fibrin in the anterior chamber of the eye
- Long-term (chronic) changes may include color variation of the iris; development of cataracts (opacity in the normally clear lens, preventing passage of light to the back part of the eye [retina]); movement of the lens out of its normal location (known as “lens luxation”); secondary glaucoma (in which the pressure within the eye [intraocular pressure] is increased secondary to inflammation in the front part of the eye); and softening and loss of tissue of the eyeball (known as “phthisis bulbi”)

CAUSES

- Infectious—fungal or mycotic infections (such as *Blastomyces*, *Cryptococcus neoformans*, *Coccidioides immitis*, *Histoplasma capsulatum*); protozoal infection (*Toxoplasma gondii*); bacterial infection (any generalized disease caused by the spread of bacteria in the blood [known as “septicemia” or “blood poisoning”]); viral infection (feline immunodeficiency virus [FIV], feline leukemia virus [FeLV], feline coronavirus); parasitic infection (due to invasion of parasitic larvae into the tissues of the eye)
- Unknown cause (so called “idiopathic disease”)—lymphocytic-plasmacytic uveitis; inflammation of the front part of the eye (including the iris) characterized by the presence of lymphocytes and plasma cells; lymphocytes are a type of white-blood cell that are formed in lymphatic tissues throughout the body; lymphocytes are involved in the immune process; plasma cells or plasmacytes are a specialized type of white-blood cell; plasma cells are lymphocytes that have been altered to produce immunoglobulin, an immune protein or antibody necessary for fighting disease
- Immune reaction to lens proteins (due to cataract or lens trauma)
- Cancer—primary tumors of the eye (such as diffuse iris melanoma, ocular sarcoma); secondary tumors (such as lymphoma) due to the spread of the cancer (metastasis)
- Metabolic—increased levels of lipids (compounds that contain fats or oils) in the blood (known as “hyperlipidemia”); increased protein in the blood leading to sludging of the blood (known as “hyperviscosity”); generalized (systemic) high blood pressure (hypertension)
- Miscellaneous—trauma; disorder of the cornea (the clear outer layer of the front of the eye) characterized by the presence of ulcers, with or without inflammation (condition known as “ulcerative keratitis”); abscess involving the cornea (known as a “corneal stromal

abscess”); presence of poisons or toxins in the blood (known as “toxemia”) of any cause

RISK FACTORS

- None specific
- Suppression of the ability to develop a normal immune response (known as “immune suppression”) and geographic location may increase incidence of certain infectious causes of inflammation of the front part of the eye, including the iris (anterior uveitis)



TREATMENT

HEALTH CARE

- Outpatient medical management generally sufficient

ACTIVITY

- No changes indicated in most cases
- Reduced exposure to bright light may alleviate discomfort

DIET

- No changes indicated

SURGERY

- None in most cases
- Specific instances requiring surgical intervention include removal of ruptured lenses and surgical management of secondary glaucoma (in which the pressure within the eye [intraocular pressure] is increased secondary to inflammation in the front part of the eye)
- Long-term (chronic) inflammation of the front part of the eye, including the iris (anterior uveitis) leading to secondary glaucoma commonly necessitates surgical removal (known as “enucleation”) of the affected eyeball(s)
- Surgical removal (enucleation) of the eyeball is recommended in cats with inflammation of the front part of the eye, including the iris (anterior uveitis), related to cancer of the iris (diffuse iris melanoma)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Steroids

- Topical steroids are medications that are applied directly to the eye, such as prednisolone acetate 1% and dexamethasone 0.1%; other topical steroids (such as betamethasone, hydrocortisone) are less effective in the treatment of inflammation within the eye—stopping topical steroids abruptly may result in rebound of inflammation of the eye
- Subconjunctival steroids are medications that are administered by injection into the moist tissues surrounding the eye (known as the “conjunctiva”), such as triamcinolone acetate and methylprednisolone; often not required; may be used in severe cases, followed by topical and/or systemic anti-inflammatory drugs
- Systemic steroids are medications that are administered by injection or by mouth (orally), such as prednisone; should only be used if generalized (systemic) infections have been eliminated as possible cause of the inflammation of the front of the eye, including the iris (anterior uveitis)

Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

- Topical NSAIDs are medications applied directly to the eye, such as flurbiprofen and diclofenac
- Systemic NSAIDs are medications administered by injection or by mouth (orally); an example is aspirin (aspirin should not be administered at the same time as systemic steroids and should be avoided if blood is present in the front of the eye [hyphema])

Topical Mydriatic/Cycloplegic Medication (to dilate the pupil and to decrease pain in the eye)

- Atropine sulfate 1%—applied directly to the eye to dilate the pupil and to decrease pain in the eye; atropine is very bitter and if cat gets medication in its mouth, excessive drooling will be seen; ointment is preferred over solution in cats, as it causes less drooling



FOLLOW-UP CARE

PATIENT MONITORING

- Recheck in 3 to 7 days, depending on severity of disease

- Pressure within the eye (intraocular pressure or IOP) should be monitored at recheck to detect secondary glaucoma (in which the pressure within the eye is increased secondary to inflammation in the front part of the eye)
- Frequency of subsequent rechecks dictated by severity of disease and response to treatment

POSSIBLE COMPLICATIONS

- Generalized (systemic) complications occur as a result of the systemic cause of the inflammation of the front of the eye, including the iris (anterior uveitis)
- Complications involving the eye include secondary glaucoma (in which the pressure within the eye [intraocular pressure or IOP] is increased secondary to inflammation in the front part of the eye)—common complication of long-term (chronic) uveitis in cats; secondary cataract (opacity in the normally clear lens, preventing passage of light to the back part of the eye [retina]) development; movement of the lens out of its normal location (lens luxation); retinal detachment; and softening and loss of tissue of the eyeball (phthisis bulbi)

EXPECTED COURSE AND PROGNOSIS

- Guarded prognosis for affected eye(s)
- Depends on underlying disease and response to treatment
- Cats with treatable underlying disease (such as toxoplasmosis) are more likely to have favorable outcome for the eye than those with idiopathic lymphocytic-plasmacytic uveitis or untreatable underlying condition (such as feline infectious peritonitis [FIP], feline immunodeficiency virus [FIV])



KEY POINTS

- Potential of generalized (systemic) diseases causing signs of inflammation of the front part of the eye, including the iris (anterior uveitis); therefore, appropriate diagnostic testing is important
- In addition to symptomatic treatment of inflammation of the front part of the eye, including the iris (anterior uveitis), treatment of underlying disease (when possible) is paramount to a positive outcome
- Compliance with treatment and follow-up recommendations may reduce the likelihood of complications

ANTERIOR UVEITIS IN DOGS

(INFLAMMATION OF THE FRONT PART OF THE EYE, INCLUDING THE IRIS)



BASICS

OVERVIEW

- Inflammation of the front part of the eye, including the iris (known as “anterior uveitis”); the iris is the colored or pigmented part of the eye—it can be brown, blue, or a mixture of colors
- May be associated with coexistent inflammation of the back part of the eye, including the retina; the retina contains the light-sensitive rods and cones and other cells that convert images into signals and send messages to the brain, to allow for vision
- May involve only one eye (known as “unilateral anterior uveitis”) or both eyes (known as “bilateral anterior uveitis”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- None for most causes
- Inflammation of the front part of the eye (anterior uveitis) associated with cysts that may be free floating or attached to the iris (known as “iridociliary cysts”) in the golden retriever (so called “golden retriever uveitis”)
- Increased incidence of uveodermatologic syndrome (a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris [anterior uveitis] and coexistent inflammation of the skin [dermatitis], characterized by loss of pigment in the skin of the nose and lips) in the Siberian husky, Akita, Samoyed, and Shetland sheepdog

Mean Age and Range

- Any age may be affected
- Mean age in uveodermatologic syndrome—2.8 years; uveodermatologic syndrome is a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris (anterior uveitis) and coexistent inflammation of the skin (dermatitis), characterized by loss of pigment in the skin of the nose and lips

SIGNS/OBSERVED CHANGES in the ANIMAL

- Red eye
- Cloudy eye—due to fluid build-up in the clear part of the eye (known as “corneal edema”); cloudiness of aqueous humor (the “aqueous humor” is the transparent liquid that fills the front part of the eyeball) due to increased protein content and suspended cellular debris (condition known as “aqueous flare”); accumulation of white-blood cells in the anterior chamber of the eye (condition known as “hypopyon”)
- Painful or uncomfortable eye—signs include squinting or spasmodic blinking (known as “blepharospasm”); avoidance of light (known as “photophobia”); or rubbing the eye
- Vision loss—variable
- Discharge from the eye; usually excessive tearing, may have mucus and/or pus in the discharge
- Keratic precipitates—aggregates of inflammatory cells adhering to various areas of the inner lining of the cornea (known as “corneal endothelium”); the cornea is the clear outer layer of the front of the eye
- Development of blood vessels in the clear part of the eye (known as “corneal vascularization”)
- Small or constricted pupil, frequently resistant to medical treatment to dilate the pupil
- Swelling of the iris
- Decreased pressure within the eye (known as “intraocular pressure” [IOP]) is consistent with anterior uveitis, but is not seen in all cases
- Scar tissue between the iris and the lens of the eye (known as “posterior synechia”); the lens is the normally clear structure directly behind the iris that focuses light as it moves toward the back part of the eye (retina)
- Accumulations of white-blood cells (hypopyon), red-blood cells (known as “hyphema”), or fibrin in the anterior chamber of the eye
- Long-term (chronic) changes may include color variation of the iris; development of cataracts (opacity in the normally clear lens, preventing passage of light to the back part of the eye [retina]); movement of the lens out of its normal location (known as “lens luxation”); secondary glaucoma (in which the pressure within the eye [intraocular pressure] is increased secondary to inflammation in the front part of the eye); and softening and loss of tissue of the eyeball (known as “phthisis bulbi”)

CAUSES

- Infectious—fungal or mycotic infections (such as *Blastomyces dermatitidis*, *Cryptococcus neoformans*, *Coccidioides immitis*, *Histoplasma capsulatum*); protozoal infections (such as *Toxoplasma gondii*, *Neospora caninum*, *Leishmania donovani*); rickettsial infections (such as *Ehrlichia canis*, *Rickettsia rickettsii*); bacterial infections (such as *Leptospira*, *Bartonella*, *Brucella canis*, *Borrelia burgdorferi* [Lyme disease], and any generalized disease caused by the spread of bacteria in the blood [known as “septicemia” or “blood poisoning”]); algal infection (*Prototheca*); viral infections (such as adenovirus, canine distemper virus, rabies virus, herpes virus); parasitic infections (due to invasion of parasitic larvae into the tissues of the eye)
- Immune-mediated—reaction to lens proteins (due to cataract or lens trauma); uveodermatologic syndrome (a rare syndrome in which the animal has inflammation in the front part of the eye, including the iris [anterior uveitis] and coexistent inflammation of the skin [dermatitis], characterized by loss of pigment in the skin of the nose and lips); post-vaccinal reaction to canine adenovirus vaccine;

inflammation of blood vessels (known as “vasculitis”)

- Cancer—primary tumors of the eye; secondary tumors due to the spread of the cancer (metastasis)
- Metabolic—increased levels of lipids (compounds that contain fats or oils) in the blood (known as “hyperlipidemia”); increased protein in the blood leading to sludging of the blood (known as “hyperviscosity”); generalized (systemic) high blood pressure (hypertension)
- Miscellaneous—unknown cause (idiopathic); trauma; golden retriever uveitis; disorder of the cornea (the clear outer layer of the front of the eye) characterized by the presence of ulcers, with or without inflammation (condition known as “ulcerative keratitis”); abscess involving the cornea (known as a “corneal stromal abscess”); inflammation of the sclera, the tough white outer coating of the eye (condition known as “scleritis”); movement of the lens out of its normal location (lens luxation); dental disease or inflammation/infection of the gums and supporting structures of the teeth (known as “periodontal disease”); presence of poisons or toxins in the blood (known as “toxemia”) of any cause

RISK FACTORS

- None specific
- Suppression of the ability to develop a normal immune response (known as “immune suppression”) and geographic location may increase incidence of certain infectious causes of inflammation of the front part of the eye, including the iris (anterior uveitis)



TREATMENT

HEALTH CARE

- Outpatient medical management generally sufficient

ACTIVITY

- No changes indicated in most cases
- Reduced exposure to bright light may alleviate discomfort

DIET

- No changes indicated

SURGERY

- None in most cases
- Specific instances requiring surgical treatment include removal of ruptured lenses and surgical management of secondary glaucoma (in which the pressure within the eye [intraocular pressure] is increased secondary to inflammation in the front part of the eye)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Steroids

- Topical steroids are medications that are applied directly to the eye, such as prednisolone acetate 1% and dexamethasone 0.1%; other topical steroids (such as betamethasone, hydrocortisone) are less effective in the treatment of inflammation within the eye—stopping topical steroids abruptly may result in rebound of inflammation of the eye
- Subconjunctival steroids are medications that are administered by injection into the moist tissues surrounding the eye (known as the “conjunctiva”), such as triamcinolone acetate and methylprednisolone; often not required; may be used in severe cases, followed by topical and/or systemic anti-inflammatory drugs
- Systemic steroids are medications that are administered by injection or by mouth (orally), such as prednisone; should only be used if generalized (systemic) infections have been eliminated as possible cause of the inflammation of the front of the eye, including the iris (anterior uveitis)

Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

- Topical NSAIDs are medications applied directly to the eye, such as flurbiprofen and diclofenac
- Systemic NSAIDs are medications administered by injection or by mouth (orally); examples are carprofen, etodolac, and aspirin (NSAIDs should not be administered at the same time as systemic steroids and should be avoided if blood is present in the front of the eye [hyphema])

Topical Mydriatic/Cycloplegic Medication (to dilate the pupil and to decrease pain in the eye)

- Atropine sulfate 1%—applied directly to the eye to dilate the pupil and to decrease pain in the eye



FOLLOW-UP CARE

PATIENT MONITORING

- Recheck in 3 to 7 days, depending on severity of disease
- Pressure within the eye (intraocular pressure or IOP) should be monitored at recheck to detect secondary glaucoma (in which the pressure within the eye is increased secondary to inflammation in the front part of the eye)
- Frequency of subsequent rechecks dictated by severity of disease and response to treatment

POSSIBLE COMPLICATIONS

- Generalized (systemic) complications, including death, occur as a result of the systemic cause of the inflammation of the front of the eye, including the iris (anterior uveitis)
- Complications involving the eye include secondary cataract (opacity in the normally clear lens, preventing passage of light to the back part of the eye [retina]) development; secondary glaucoma (in which the pressure within the eye [intraocular pressure or IOP] is increased secondary to inflammation in the front part of the eye); movement of the lens out of its normal location (lens luxation); retinal detachment; and softening and loss of tissue of the eyeball (phthisis bulbi)

EXPECTED COURSE AND PROGNOSIS

- Extremely variable; depends on underlying disease and response to treatment



KEY POINTS

- Potential of generalized (systemic) diseases causing signs of inflammation of the front part of the eye, including the iris (anterior uveitis); therefore, appropriate diagnostic testing is important
- In addition to symptomatic treatment of inflammation of the front part of the eye, including the iris (anterior uveitis), treatment of underlying disease (when possible) is paramount to a positive outcome
- Compliance with treatment and follow-up recommendations may reduce the likelihood of complications

VAGINAL DISCHARGE IN DOGS



BASICS

OVERVIEW

- “Vaginal” refers to the vagina; the “vagina” is the tubular passageway leading from the opening of the vulva to the cervix of the uterus; “vulvar” refers to the vulva; the “vulva” is the external genitalia of females
- “Vaginal discharge” is any substance (such as blood, mucus, pus) coming from the vagina, through the vulvar opening
- “Bitch” is a female dog

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Mean Age and Range

- Bitches prior to going through puberty (known as “prepubertal bitches”)—anatomic abnormalities and prepubertal inflammation of the vagina (known as “prepubertal vaginitis”) more common
- Bitches in “heat” or “estrus” or following delivery of puppies (whelping)—normal vaginal discharges are common
- Bitches that recently have completed their “heat” or “estrous cycle” or are pregnant or following delivery of puppies (whelping)—vaginal discharge may be more serious

Predominant Sex

- Females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Discharge from the vulva (the external genitalia); discharge may be blood; blood, mucus, and tissue debris (known as “lochia”) following delivery of puppies; pus; urine; or feces
- Spotting
- Scooting
- Attracting males
- Delivering puppies (whelping or parturition)—with postpartum discharge
- History of “heat” or “estrus” during the preceding 2 months—vaginal discharge may be related to inflammation with accumulation of pus in the uterus (known as “pyometra”)

CAUSES

Discharge Containing Serum and Blood (known as “serosanguineous discharge”)

- Normal during early heat cycle (known as “proestrus”) and sometimes into “heat” or “estrus”
- Urinary tract infection
- Foreign body
- Tumor or cancer of the vagina—such as transmissible venereal tumor; leiomyoma
- Vaginal trauma
- Fetal death
- Localized accumulation of blood in the vagina (known as a “vaginal hematoma”)
- Tumor or cancer of the ovaries
- Blood-clotting disorders (known as “coagulopathies”)

Discharge Containing Blood, Mucus, and Tissue Debris (Lochia) and Other Discharges Following Birth (Postpartum)

- Normal postpartum discharge—for 6 to 8 weeks
- Subinvolution of placental sites (condition in which the placental attachment sites in the lining of the uterus do not return to normal in 6 to 8 weeks)—discharge lasting longer than 8 weeks
- Retained placentas or afterbirth (condition in which one or more placentas remains within the uterus following the delivery of the puppy or puppies)
- Inflammation of the lining of the uterus (known as “metritis”)

Discharge Containing Pus

- Normal in early diestrus (“diestrus” is the phase of the “heat” cycle immediately after “standing heat;” slight amount of discharge)
- Inflammation of the vagina in a bitch prior to puberty (prepubertal vaginitis)
- Primary inflammation of the vagina (vaginitis)
- Secondary inflammation of the vagina (vaginitis)—from anatomic abnormality, foreign body, urinary tract infection, enlargement of the clitoris (known as “clitoral hypertrophy”), vaginal tumor, and fetal death
- Inflammation with accumulation of pus in the uterus (pyometra)
- Embryonic and fetal death
- Inflammation of the lining of the uterus following birth of the puppies (postpartum metritis)
- Inflammation of the skin around the vulva (known as “perivulvar dermatitis”)
- Zinc toxicity—reported

Other

- Urine or feces—with congenital (present at birth) anatomic abnormalities
- Acquired (condition that develops sometime later in life/after birth) inflammation of the skin around the vulva (perivulvar dermatitis) also can be mistaken for vaginal discharge
- Urine from ectopic ureters or lack of control of urination (incontinence) from low levels of estrogen (known as “hypoestrogenism”); the ureters are tubes from the kidneys to the bladder; during development, they may not attach to the bladder properly or may attach to reproductive organs instead; when this occurs, they are called “ectopic ureters” and one or both can terminate in the lower urethra, uterus, or vagina
- Normal mucous discharge during pregnancy

RISK FACTORS

- Medications or products containing male hormones (androgens)—may cause enlargement of the clitoris (clitoral hypertrophy)
- Prophylactic antibiotics—may alter the normal vaginal bacteria and allow overgrowth of disease-causing species
- Estrogen medications given during certain phases of the “heat” or “estrous” cycle—predispose patient to inflammation with accumulation of pus in the uterus (pyometra)



TREATMENT

HEALTH CARE

- Outpatient, unless inflammation of the lining of the uterus (metritis) or inflammation with accumulation of pus in the uterus (pyometra) is noted and spaying (ovariohysterectomy) may be indicated
- Supportive fluids—for inflammation of the lining of the uterus (metritis) or inflammation with accumulation of pus in the uterus (pyometra), if the patient is ill
- Medical treatment for inflammation with accumulation of pus in the uterus (pyometra)—performed in a hospital and with great care
- Remove or treat any inciting cause—foreign body; tumor; anatomic abnormality; urinary tract infection; medications or products containing male hormones (androgens) or estrogens
- Inflammation of the vagina in a bitch prior to puberty (prepubertal vaginitis)—usually resolves spontaneously after the first “heat” or “estrus”
- Subinvolution of placental sites (condition in which the placental attachment sites in the lining of the uterus do not return to normal in 6 to 8 weeks)—rarely requires treatment

SURGERY

- Depends on cause of vaginal discharge
- Bitches with inflammation of the lining of the uterus (metritis) or inflammation with accumulation of pus in the uterus (pyometra) may require surgery; a “spay” or “ovariohysterectomy” (in which the ovaries and uterus are removed surgically) may be indicated
- Bitches with cancer or congenital (present at birth) anatomic abnormalities may need surgery



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Inflammation of the lining of the uterus (metritis)—systemic antibiotics, if the patient is ill
- Inflammation of the vagina in a bitch prior to puberty (prepubertal vaginitis)—diethylstilbestrol to induce “heat” or “estrus” may help; long-term effects not documented; discuss the risks and benefits of treatment with your pet’s veterinarian
- Primary inflammation of the vagina (vaginitis)—systemic antibiotics; vaginal douches, as directed by your pet’s veterinarian
- Inflammation with accumulation of pus in the uterus (pyometra)—prostaglandin (substance that stimulates the uterus) and systemic antibiotics, if the patient is not extremely ill
- Transmissible venereal tumor—vincristine, a chemotherapy drug



FOLLOW-UP CARE

PATIENT MONITORING

- Ultrasonography or X-rays—determine size and contents of the uterus in cases with inflammation of the lining of the uterus (metritis) or inflammation with accumulation of pus in the uterus (pyometra)

PREVENTIONS AND AVOIDANCE

- Depends on cause of vaginal discharge
- Spaying (ovariohysterectomy) will prevent inflammation of the lining of the uterus (metritis) or inflammation with accumulation of pus in the uterus (pyometra)

POSSIBLE COMPLICATIONS

- Toxic shock—with severe inflammation of the lining of the uterus (metritis) or inflammation with accumulation of pus in the uterus (pyometra)

EXPECTED COURSE AND PROGNOSIS

- Depends on cause of vaginal discharge



KEY POINTS

- Vaginal discharge is any substance (such as blood, mucus, pus) coming from the vagina, through the vulvar opening

INFLAMMATION OF THE VAGINA (VAGINITIS)



BASICS

OVERVIEW

- “Vaginal” refers to the vagina; the “vagina” is the tubular passageway leading from the opening of the vulva to the cervix of the uterus; “vulvar” refers to the vulva; the “vulva” is the external genitalia of females
- “Vaginitis” is inflammation of the vagina or vestibule (space at the entrance of the vagina)
- “Bitch” is a female dog

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Primarily dogs

Mean Age and Range

- Anatomic abnormalities and inflammation of the vagina in animals prior to puberty (known as “prepubertal vaginitis”)—suspect in bitches that have not gone through puberty
- May occur at any age in any breed or with any ovarian status (that is, whether intact or spayed)

Predominant Sex

- Females

SIGNS/OBSERVED CHANGES in the ANIMAL

- Discharge from the vulva
- Frequent voiding of small volumes of urine (known as “pollakiuria”)
- Vaginal licking
- Spotting
- Scooting
- Attracting males
- Discharge from the vagina; vaginal discharge is any substance (such as blood, mucus, pus) coming from the vagina, through the vulvar opening
- Possibly inflamed vulva and vagina

CAUSES

- Immature vagina (prior to puberty)
- Foreign bodies
- Urinary tract infections
- Vaginal trauma
- Urine or feces contamination in patients with certain congenital (present at birth) anatomic abnormalities
- Urine contamination in patients with ectopic ureters; the ureters are tubes from the kidneys to the bladder; during development, they may not attach to the bladder properly or may attach to reproductive organs instead; when this occurs, they are called “ectopic ureters” and one or both can terminate in the lower urethra, uterus, or vagina
- Inability to control urination (known as “incontinence”) owing to low levels of estrogen (known as “hypoestrogenism”)
- Tumor or cancer of the vagina—such as transmissible venereal tumor; leiomyoma
- Bacterial infections, such as *Pasteurella*; *Streptococcus*; *E. coli*; *Pseudomonas*; *Mycoplasma*; *Chlamydia*; *Brucella canis*
- Viral infection—herpes
- Localized accumulation of blood in the vagina (known as a “vaginal hematoma”)
- Vaginal abscess
- Medications or products containing male hormones (known as “androgens”)
- Narrowing of the entrance to the vagina (known as a “vestibulovaginal stricture”)
- Zinc toxicity reported

RISK FACTORS

- Medications or products containing male hormones (androgens)—may cause enlargement of the clitoris (known as “clitoral hypertrophy”)
- Prophylactic antibiotics—may alter the normal vaginal bacteria and allow overgrowth of disease-causing species
- Anatomic abnormalities in bitches prior to going through puberty



TREATMENT

HEALTH CARE

- Usually treated as outpatients
- Inpatient—surgical management of anatomic abnormalities, foreign bodies, or tumors/cancer

ACTIVITY

- Normal

DIET

- Normal

SURGERY

- Remove or treat any inciting causes—foreign body; tumor or cancer; anatomic abnormalities
- Surgical removal of the vagina (known as “vaginectomy”)—has been used in patients that do not respond to medical treatment



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Inflammation of the Vagina Prior to Puberty (Prepubertal Vaginitis)

- Inflammation of the vagina in a bitch prior to puberty (prepubertal vaginitis)—diethylstilbestrol to induce “heat” or “estrus” may help; long-term effects not documented; discuss the risks and benefits of treatment with your pet’s veterinarian

Primary Inflammation of the Vagina (Vaginitis)

- Appropriate systemic antibiotics—normally eradicate susceptible bacteria within 24 hours
- Vaginal douches, as directed by your pet’s veterinarian—0.05% chlorhexidine or 0.5% povidone-iodine twice daily until the discharge resolves; reported to be beneficial



FOLLOW-UP CARE

PATIENT MONITORING

- Inflammation of the vagina in an animal prior to puberty (prepubertal vaginitis)—re-examine after the first “heat” or “estrus” or when physical maturity is reached
- Mature patients—re-examine after a 14-day course of antibiotics
- If condition persists—reevaluate for an underlying or another cause; perform a vaginal bacterial culture and sensitivity test

PREVENTIONS AND AVOIDANCE

- Some rationale may be considered for delaying spaying (ovariohysterectomy) until after the first “heat” or “estrus” in patients prior to puberty with long-term (chronic) inflammation of the vagina (vaginitis), because some cases do not respond to medical treatment after the patient is spayed

EXPECTED COURSE AND PROGNOSIS

- Inflammation of the vagina in an animal prior to puberty (prepubertal vaginitis)—normally resolves after the first “heat” or “estrus”

- Adults—inflammation of the vagina (vaginitis) usually resolves if the causative factor is removed; antibiotic therapy and vaginal douches may hasten recovery of uncomplicated, long-term (chronic) cases to within 2 weeks



KEY POINTS

- Inflammation of the vagina in an animal prior to puberty (prepubertal vaginitis) normally resolves after the first “heat” or “estrus” and antibiotic therapy is not needed
- Inflammation of the vagina (vaginitis) in adults often is associated with a correctable predisposing factor
- Spaying (ovariohysterectomy) and isolation of patients should be considered for patients infected with *Brucella canis*
- Medications or products containing male hormones (androgens) or estrogens must be discontinued, as directed by your pet’s veterinarian

IRREGULAR HEART BEAT: VENTRICULAR PREMATURE COMPLEXES



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- An electrocardiogram (“ECG”) is a recording of the electrical impulse activity of the heart; the normal ECG is a tracing with P, QRS, and T waves; the P wave is the first upward deflection of the ECG tracing that looks like a “bump” in the tracing; the P waves are a measure of the electrical activity of the atria; the QRS looks like an exaggerated “W” with the Q wave being a short, downward deflection, the R being a tall, spiked upward deflection, and the S being another short, downward deflection; the QRS is a measure of the electrical activity of the ventricles; finally the T wave may be an upward or downward deflection of the ECG tracing; the T wave is a measure of ventricular recovery prior to the next contraction
- “Ventricular premature complexes” are a type of irregular heart beat; an electrical impulse is initiated within the ventricles instead of the sinoatrial (SA) node, causing the ventricles to contract too early (thus the “premature”)
- Recording of an electrocardiogram (“ECG,” a recording of the electrical activity of the heart), characterized by abnormal QRS complexes not associated with P waves
- Also known as “VPCs”

GENETICS

- Inherited ventricular irregular heart beat (known as an “arrhythmia”) in German shepherd dogs

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predisposition

- Common in large-breed dogs with disease of the heart muscle (known as “cardiomyopathy”), especially boxers and Doberman pinschers
- Inherited ventricular arrhythmia in German shepherd dogs
- Common in cats with disease of the heart muscle (cardiomyopathy); occasionally seen in cats with excessive levels of thyroid hormone (known as “hyperthyroidism”)

Mean Age and Range

- Seen in all age groups

SIGNS/OBSERVED CHANGES in the ANIMAL

- Weakness
- Exercise intolerance
- Fainting (known as “syncope”)
- Sudden death
- Often no signs observed
- Irregular heart beats associated with pulse deficits (situation in which the number of heart beats and number of pulses do not match, usually indicating inadequate filling of the ventricles prior to contraction); may hear splitting of the first or second heart sound when listening to the heart with a stethoscope
- Heart beats may be normal during physical examination, if the irregular heart beat (arrhythmia) is intermittent
- May observe signs of congestive heart failure (such as cough and difficulty breathing [known as “dyspnea”]) or detect a heart murmur, depending on the cause of irregular heart beat; congestive heart failure is a condition in which the heart cannot pump an adequate

volume of blood to meet the body's needs

CAUSES

- Disease of heart muscle (cardiomyopathy)
- Congenital (present at birth) heart defects (especially subaortic stenosis, a birth defect involving narrowing just below the aortic valve, the heart valve from the left ventricle to the aorta [the main artery of the body])
- Long-term (chronic) disease of the heart valves
- Stomach dilates with gas and/or fluid (known as “gastric dilatation”), and subsequently rotates around its short axis (known as “volvulus”)—condition known as “gastric dilatation-volvulus” or “bloat”
- Traumatic inflammation of the heart muscle (known as “traumatic myocarditis”) in dogs
- Digitalis toxicity; digitalis is a heart medication
- Excessive levels of thyroid hormone (hyperthyroidism) in cats
- Heart tumors or cancer
- Inflammation of the heart muscle (myocarditis)
- Inflammation of the pancreas (known as “pancreatitis”)

RISK FACTORS

- Low levels of potassium in the blood (known as “hypokalemia”)
- Low levels of magnesium in the blood (known as “hypomagnesemia”)
- Acid–base disturbances (abnormalities in blood pH levels)
- Low levels of oxygen in the blood and tissues (known as “hypoxia”)



TREATMENT

HEALTH CARE

- Generally outpatient basis
- Varies with underlying cause
- Correct any low levels of potassium in the blood (hypokalemia) or low levels of magnesium in the blood (hypomagnesemia)

ACTIVITY

- Restrict if the irregular heart beat is accompanied by clinical signs or evidence of structural heart disease

DIET

- Mild to moderate sodium restriction if patient is in congestive heart failure; congestive heart failure is a condition in which the heart cannot pump an adequate volume of blood to meet the body's needs

SURGERY

- Continuous recording of an electrocardiogram (“ECG,” a recording of the electrical activity of the heart) recommended while patient is anesthetized
- Premedicating the patient with acepromazine raises the threshold (and thus decreases the likelihood) for ventricular fibrillation (a condition in which the ventricles rapidly contract in a chaotic manner and cannot pump blood to the body—it is a life-threatening emergency)
- Mask inductions of anesthesia are not recommended; mask induction can aggravate the irregular heart beat (arrhythmia)
- Avoid medications used to increase the heart rate (known as “anticholinergics”) during anesthesia and surgery unless slow heart rate (known as “bradycardia”) develops



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drug therapy in the absence of clinical signs—controversial; studies in people with ventricular premature complexes that do not have

symptoms and sudden lack of blood supply to the heart muscle that leads to death of tissues (known as “myocardial infarctions”) demonstrated a high incidence of sudden death when treatment is initiated with medications to control the irregular heart beats (known as “class I antiarrhythmic agents”); no similar studies have been done in veterinary patients

- Medications to control the irregular heart beats (antiarrhythmic drugs) generally are not prescribed unless the animal has evidence of low blood volume being pumped by the heart (known as “low cardiac output”), such as episodic weakness or fainting (syncope) or the patient is at high risk of sudden death

- If medications to control the irregular heart beat (antiarrhythmic treatment) is initiated in an attempt to lower the risk of sudden death, a β -blocker or sotalol generally is selected; no studies have been done to confirm effectiveness of β -blockers for prevention of sudden death in dogs or cats

Dogs

- Patient not in congestive heart failure or does not have low blood pressure (known as “hypotension”)—treatment with a β -blocker (such as propranolol, atenolol, or metoprolol)
- Patient in congestive heart failure or has low blood pressure (hypotension)—treatment with a medication to control the irregular heart beat (class I antiarrhythmic agent, such as mexiletine or procainamide)
- Combine a medication to control the irregular heart beat (class I antiarrhythmic drug) with a β -blocker, if significant irregular heart beats persist
- Consider sotalol or amiodarone for irregular heart beats that do not respond to medical treatment

Cats

- β -blocker—atenolol
- Consider sotalol or procainamide for cats that do not tolerate β -blockers



FOLLOW-UP CARE

PATIENT MONITORING

- Holter monitoring (where the patient wears a “vest” in which a continuous, mobile battery-powered ECG monitor has been placed; the ECG recording is performed over several hours, giving a better overall picture of the heart rate and rhythm) is preferred for monitoring severity of the irregular heart beat (arrhythmia) and effectiveness of treatment; the goal of treatment is to reduce the frequency of ventricular premature complexes by more than 80%

- Serial recordings of an electrocardiogram (“ECG,” a recording of the electrical activity of the heart) are not as useful as Holter monitoring—ventricular premature complexes and sudden onset of a very fast heart rate originating in the ventricles, causing the heart to beat ineffectively (known as “paroxysmal ventricular tachycardia”) can occur sporadically through the day

- Serum digoxin levels should be monitored in patients receiving this heart medication

PREVENTIONS AND AVOIDANCE

- Correct factors that increase the risk of ventricular premature complexes, such as low levels of potassium in the blood (hypokalemia), low levels of magnesium in the blood (hypomagnesemia), low levels of oxygen in the heart muscle (known as “myocardial hypoxia”), and digoxin toxicity

POSSIBLE COMPLICATIONS

- Fainting (syncope)
- Sudden death

EXPECTED COURSE AND PROGNOSIS

- If cause is metabolic—condition may resolve with good prognosis
- If condition is associated with heart disease—prognosis is guarded; ventricular premature complexes may increase the risk of sudden death



KEY POINTS

- “Ventricular premature complexes” are a type of irregular heart beat; an electrical impulse is initiated within the ventricles instead of the sinoatrial (SA) node, causing the ventricles to contract too early
- Potential for the irregular heart beat (arrhythmia) to worsen and for the animal to have signs of fainting (syncope) or sudden death

VENTRICULAR SEPTAL DEFECT



BASICS

OVERVIEW

- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles
- “Ventricular septal defect” is a type of heart birth defect due to an abnormal communication between the two ventricles
- One of the most common congenital (present at birth) heart malformations in cats; less common in dogs
- Also known as “VSD”

GENETICS

- Greater likelihood of having ventricular septal defect is recognized in certain breeds; no genetic transmission has been established

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilections

- English bulldog, English springer spaniel, basset hound, Akita, West Highland white terrier, Lakeland terrier

Mean Age and Range

- Most defects detected during routine examination of puppies and kittens

SIGNS/OBSERVED CHANGES in the ANIMAL

- Usually no clinical signs
- Heart murmur or abnormal heart sounds may be heard when listening to the heart with a stethoscope
- Femoral pulses usually are normal
- Gums and moist tissues of the body (known as “mucous membranes”)—usually pink, unless high blood pressure in the lungs (known as “pulmonary hypertension”) causes a right-to-left flow of blood through the ventricular septal defect, leading to decreased oxygen in the blood (known as “hypoxemia”)
- May have signs of left-sided congestive heart failure, such as exercise intolerance, fainting (known as “syncope”), and cough; “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs
- Rapid heart rate (known as “tachycardia”), difficulty breathing (known as “dyspnea”), and short, rough snapping sounds (known as “crackles”) heard when listening to the heart with a stethoscope may be evident if left-sided congestive heart failure occurs

CAUSES

- Congenital (present at birth) disease; may have a genetic basis



TREATMENT

HEALTH CARE

- Clinical signs are related to congestive heart failure (in which the heart cannot pump an adequate volume of blood to meet the body’s needs); most patients can be treated as outpatients

ACTIVITY

- Restrict if animal has congestive heart failure (in which the heart cannot pump an adequate volume of blood to meet the body’s needs)
- No need to restrict activity in pets without clinical signs (known as “asymptomatic patients”) with small ventricular septal defects

DIET

- Moderate sodium restriction recommended for pets with congestive heart failure (in which the heart cannot pump an adequate volume of blood to meet the body’s needs)

SURGERY

- Consider surgical repair of the ventricular septal defect; requires heart-lung (cardiopulmonary) bypass for defects associated with a large shunt—cardiopulmonary bypass presently is performed at a small number of veterinary centers
- Consider pulmonary artery banding as a means to control signs and to improve the patient's condition, but not to cure the ventricular septal defect (known as a "palliative procedure") for patients with moderate or large shunts and congestive heart failure (in which the heart cannot pump an adequate volume of blood to meet the body's needs)
- Transcatheter correction of ventricular septal defect has been reported; the therapeutic role of this technique is yet to be determined



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Treatment of pets with congestive heart failure (in which the heart cannot pump an adequate volume of blood to meet the body's needs)—medications to remove excess fluid from the body (known as "diuretics," such as furosemide) and heart medications (such as enalapril and digoxin)



FOLLOW-UP CARE

PATIENT MONITORING

- Periodic echocardiographic (use of ultrasound to evaluate the heart and major blood vessels) or X-ray evaluation suggested for patients without clinical signs

PREVENTIONS AND AVOIDANCE

- Breeding affected animals is not recommended

POSSIBLE COMPLICATIONS

- Left-sided congestive failure (in which the heart cannot pump an adequate volume of blood to meet the body's needs)
- Bacterial infection of the lining of the heart (known as "bacterial endocarditis")
- High blood pressure in the lungs (pulmonary hypertension)
- Irregular heart beats (known as "arrhythmias")

EXPECTED COURSE AND PROGNOSIS

- Patients with small shunts may have a normal life span; some ventricular septal defects may not cause clinical signs
- Coexistent heart abnormalities worsen the prognosis
- Patients with obvious congestive heart failure (in which the heart cannot pump an adequate volume of blood to meet the body's needs) may live 6 to 18 months with medical treatment
- The development of high blood pressure in the lungs (pulmonary hypertension) and reversal of blood flow (right to left through the ventricular septal defect) is uncommon



KEY POINTS

- Definitive surgical correction of ventricular septal defect is not widely available
- If congestive heart failure (in which the heart cannot pump an adequate volume of blood to meet the body's needs) develops, the condition is terminal, even with medical care
- Patients with obvious congestive heart failure may live 6 to 18 months with medical treatment

VENTRICULAR TACHYCARDIA



BASICS

OVERVIEW

- “Ventricular” refers to the ventricles of the heart; “tachycardia” is the medical term for rapid heart rate
- The heart of the dog or cat is composed of four chambers; the top two chambers are the right and left atria and the bottom two chambers are the right and left ventricles; heart valves are located between the right atrium and the right ventricle (tricuspid valve); between the left atrium and the left ventricle (mitral valve); from the right ventricle to the main pulmonary (lung) artery (pulmonary valve); and from the left ventricle to the aorta (the main artery of the body; valve is the aortic valve)
- In order to pump blood to the lungs and body, the heart must work in a coordinated fashion; the normal control or “pacemaker” of the heart is the sinus or sinoatrial (SA) node, which starts the electrical impulse to begin the coordinated contraction of the heart muscles—the electrical impulse causes the atria to contract, pumping blood into the ventricles; the electrical impulse moves through the atrioventricular (AV) node and into the ventricles, causing the ventricles to contract and to pump blood to the lungs (right ventricle) and the body (left ventricle)
- The normal heart rate for dogs varies based on the size of the dog; however, the general range is 60 to 180 beats per minute (with smaller dogs have faster normal heart rates)
- The general range for normal heart rate in cats is 120 to 240 beats per minute
- “Ventricular tachycardia” (VT) is a rapid heart rate; it may occur in structurally normal hearts (hereditary irregular heart beats [known as “arrhythmias”]) or may be a consequence of abnormalities of heart muscle associated with cardiomyopathy (disease of the heart muscle), significant heart-valve disease or inflammation of the heart muscle (known as “myocarditis”)
- Ventricular tachycardia is a common cause of irregular heart beats (arrhythmias) in dogs; uncommon in cats
- An electrocardiogram (“ECG”) is a recording of the electrical impulse activity of the heart; the normal ECG is a tracing with P, QRS, and T waves; the P waves are the first upward deflection of the ECG tracing that look like a “bump” in the tracing; the P waves are a measure of the electrical activity of the atria; the QRS looks like an exaggerated “W” with the Q wave being a short, downward deflection, the R being a tall, spiked upward deflection, and the S being another short, downward deflection; the QRS is a measure of the electrical activity of the ventricles; finally the T wave may be an upward or downward deflection of the ECG tracing; the T wave is a measure of ventricular recovery prior to the next contraction
- Ventricular tachycardia is diagnosed by changes in the electrocardiogram (ECG), including three or more abnormal beats that begin in the ventricle and causes the heart muscle to contract too quickly (known as “ventricular premature contractions”) in a row; if P waves are visible, they are not associated with the QRS complexes; QRS complexes typically are wide and bizarre in appearance
- Rapid ventricular heart rate (ventricular tachycardia) may be intermittent (paroxysmal) or sustained
- Certain breeds (boxers, Doberman pinschers, and German shepherd dogs) have characteristic, breed-specific electrocardiogram (ECG) changes

GENETICS

- Heart-muscle disease of the right ventricle (known as “arrhythmogenic right ventricular cardiomyopathy” or “ARVC”) in boxers and heart-muscle disease characterized by a weak, flabby heart and ventricular tachycardia (known as “dilated cardiomyopathy with ventricular tachycardia”) in Doberman pinschers are both inherited as autosomal dominant traits
- Ventricular irregular heart beats (arrhythmias) and sudden cardiac death are hereditary in German shepherd dogs; mode of inheritance involves multiple genes (known as a “polygenic trait”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predilection

- Commonly seen in large-breed dogs with heart-muscle disease (cardiomyopathy), especially boxers and Doberman pinschers
- German shepherd dogs with sudden cardiac death

Mean Age and Range

- All age groups, if not breed-specific ventricular tachycardia
- Boxers with heart-muscle disease of the right ventricle (arrhythmogenic right ventricular cardiomyopathy) usually present to the veterinarian at 4 to 6 years of age, frequency and severity of the irregular heart beat (arrhythmia) usually increases over time
- Doberman pinschers with “hidden” heart-muscle disease (known as “occult cardiomyopathy”) typically develop ventricular irregular heart beats (arrhythmias) beginning at 3 to 6 years of age, but ventricular arrhythmias can occur much later in life; frequency and severity of the arrhythmia usually increases over time

- German shepherd dogs develop ventricular irregular heart beats (arrhythmias) at 12 to 16 weeks of age; the frequency and severity of arrhythmias increases until 24 to 30 weeks of age; after 8 months of age, the arrhythmia severity stabilizes or starts to decrease

SIGNS/OBSERVED CHANGES in the ANIMAL

- May have no clinical signs
- Fainting (known as “syncope”)
- Weakness
- Exercise intolerance
- Sudden death
- Rapid heart rate (tachycardia)
- Femoral pulses may vary or be weak
- Heart murmur may be present
- Signs of congestive heart failure may be present; signs include cough; difficulty breathing (known as “dyspnea”); bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red-blood cells (condition known as “cyanosis”); “congestive heart failure” is a condition in which the heart cannot pump an adequate volume of blood to meet the body’s needs

CAUSES

- Heart-muscle disease (cardiomyopathy)
- Congenital (present at birth) heart defects (especially subaortic stenosis, a birth defect involving narrowing of the area just below the aortic valve, the heart valve from the left ventricle to the aorta [the main artery of the body])
- Long-term (chronic) heart-valve disease
- Condition in which the stomach dilates with gas and/or fluid (known as “gastric dilatation”), and subsequently rotates around its short axis (known as “volvulus”)—condition known as “gastric dilatation-volvulus” or “bloat”
- Traumatic inflammation of the heart muscle (myocarditis) in dogs
- Digitalis toxicity; digitalis is a heart medication
- Increased levels of thyroid hormone (known as “hyperthyroidism”) in cats
- Heart tumors or cancer
- Inflammation of the heart muscle (myocarditis)
- Inflammation of the pancreas (known as “pancreatitis”)

RISK FACTORS

- Low levels of potassium in the blood (known as “hypokalemia”) or high levels of potassium in the blood (known as “hyperkalemia”)
- Low levels of magnesium in the blood (known as “hypomagnesemia”)
- Acid–base disturbances
- Low levels of oxygen in the blood (known as “hypoxemia”)
- Cancer (such as hemangiosarcoma of the heart or spleen)



TREATMENT

HEALTH CARE

- Varies with underlying cause
- Most patients with intermittent ventricular tachycardia can be worked up safely to check for underlying diseases (diagnostics may include electrocardiogram [“ECG,” a recording of the electrical activity of the heart], echocardiogram [use of ultrasound to evaluate the heart and major blood vessels], and blood work) and establish a true baseline of the quantity and quality of the irregular heart beats (arrhythmias) by a 24-hour Holter monitoring (where the patient wears a “vest” in which a continuous, mobile battery-powered ECG monitor has been placed; the ECG recording is performed over several hours, giving a better overall picture of the heart rate and rhythm)
- Correct any low levels of potassium in the blood (hypokalemia) or low levels of magnesium in the blood (hypomagnesemia), if possible, prior to instituting medical therapy
- If an animal is unstable (such as being unable to stand, weak or has frequent fainting [syncope]), immediate intravenous treatment in a hospital setting with continuous ECG monitoring may be required
- Once the irregular heart beat (arrhythmia) is controlled and the patient is stable, oral medication should be instituted

- To date, no medical treatment is available to prevent sudden death in animals afflicted with rapid, irregular ventricular heart beats (known as “ventricular tachyarrhythmias”)

ACTIVITY

- Generally speaking, no benefit has been demonstrated to restrict exercise
- Boxers tend to have an increased incidence of rapid ventricular heart beats (ventricular tachycardia) during excitement, so owners should know what specific situations to avoid to prevent excitement (if possible)

SURGERY

- When possible, determine the cause of the irregular heart beats (arrhythmias) and treat it prior to inducing general anesthesia for any reason
- Avoid using medications that may worsen the irregular heart beats (arrhythmias), such as xylazine, medetomidine, or thiopental
- Mask inductions for anesthesia are not recommended in inadequately sedated patients with irregular ventricular heart beats (ventricular arrhythmias)
- Continuous electrocardiogram (ECG, a recording of the electrical activity of the heart) monitoring while patient is anesthetized



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

Dogs

- Sudden (acute) life-threatening, rapid, irregular ventricular heart beats (ventricular tachycardia)—slowly administer lidocaine into the vein (intravenous administration) to convert to a normal heart rhythm (known as “sinus rhythm”); follow with lidocaine continuous rate intravenous infusion
- If ventricular tachycardia does not respond to lidocaine—slowly administer procainamide into the vein to convert to a normal heart rhythm (sinus rhythm); follow with procainamide infusion
- If the patient does not respond to lidocaine or procainamide, slowly administer esmolol (a short-acting β -blocker) into the vein or as a constant rate infusion
- Long-term (chronic) rapid, irregular ventricular heart beats (ventricular tachycardia) in a stable patient—sotalol may be administered or a combination of mexiletine with a β -blocker (such as atenolol or sotalol) may be more effective for ventricular tachycardia that is not responsive to medical treatment (known as “refractory ventricular tachycardia”), especially in boxers
- Combination of mexiletine and sotalol is most effective in German shepherd dogs
- Consider amiodarone for ventricular tachycardia that is not responsive to medical treatment (refractory ventricular tachycardia); amiodarone is slowly effective and it has several potential side effects that you should discuss with your dog’s veterinarian

Cats

- Use lidocaine cautiously and only for sustained rapid, irregular ventricular heart beats (ventricular tachycardia), as seizures are a common side effect of lidocaine in cats
- Atenolol is the preferred treatment in cats
- Consider sotalol for ventricular tachycardia that is not responsive to medical treatment (refractory ventricular tachycardia)



FOLLOW-UP CARE

PATIENT MONITORING

- Holter monitoring (where the patient wears a “vest” in which a continuous, mobile battery-powered ECG monitor has been placed; the ECG recording is performed over several hours, giving a better overall picture of the heart rate and rhythm) is preferred for monitoring severity of the rapid, irregular ventricular heart beat (ventricular tachycardia) and effectiveness of treatment
- Follow-up 24-hour Holter monitoring is required to test effectiveness of treatment to control the irregular heart beat (arrhythmia)
- Serial electrocardiograms (ECGs, recordings of the electrical activity of the heart) and telemetry (heart rate and rhythm recordings transmitted to a centralized monitor using radio waves) can be used—not as useful as Holter monitoring because abnormal beats that begin in the ventricle and causes the heart muscle to contract too quickly (ventricular premature contractions) and intermittent rapid ventricular heart rate (paroxysmal ventricular tachycardia) can occur sporadically through the day
- Patients receiving digoxin (a heart medication)—serum digoxin levels should be measured after one week of treatment; obtain blood

sample for testing 8 to 10 hours post pill

- Dogs treated with amiodarone—serial blood tests (serum chemistries) are recommended, since increases in liver enzyme activities usually precede onset of clinical signs of amiodarone toxicity; liver enzymes should be measured after 7 days of drug loading and once monthly during maintenance therapy

PREVENTIONS AND AVOIDANCE

- Correct predisposing factors (such as low levels of potassium in the blood [hypokalemia], low levels of magnesium in the blood [hypomagnesemia], low levels of oxygen in the heart muscle [known as “myocardial hypoxia”], and digoxin toxicity)

POSSIBLE COMPLICATIONS

- Fainting (syncope)
- Sudden death

EXPECTED COURSE AND PROGNOSIS

- If cause is metabolic—condition may resolve with a good prognosis
- If condition is associated with heart disease—prognosis guarded, because underlying heart disease is likely long-term (chronic) and progressive and therefore, the irregular heart beats (arrhythmias) may worsen too over time; presence of significant rapid, irregular ventricular heart beat (ventricular tachycardia) increases risk of sudden death
- Approximately 50% of German shepherd dogs with more than 10 runs of rapid, irregular ventricular heart beats (ventricular tachycardia) per 24 hours die suddenly; if dogs reach 18 months of age, the probability of sudden death decreases
- Unlike boxers with heart-muscle disease of the right ventricle (arrhythmogenic right ventricular cardiomyopathy), Doberman pinschers with heart-muscle disease characterized by a weak, flabby heart and ventricular tachycardia (dilated cardiomyopathy with ventricular tachycardia) may die suddenly during their first fainting (syncopal) episode



KEY POINTS

- Potential for sudden death

VESTIBULAR DISEASE IN CATS



BASICS

OVERVIEW

- Sudden (acute) nonprogressive disturbance of the peripheral vestibular system in cats
- The vestibular system controls the animal's sense of equilibrium, balance, and orientation; it is composed of the inner ear, nerves, and brain
- Cause for vestibular disease in the cat is unknown (so called "idiopathic vestibular disease")

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Cats

Mean Age and Range

- Any age; rarely observed in cats less than 1 year of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden onset of severe disorientation, falling, rolling, leaning, vocalizing, and crouched posture with tendency to panic when picked up
- Head tilt—always toward the side of the nervous system lesion; occasionally wide, side-to-side movements of the head
- Irregular eye movements (known as "nystagmus")
- Wobbly, incoordinated or "drunken" appearing gait or movement (known as "ataxia") with tendency to roll and fall toward the side of the head tilt
- Strength is normal
- May be reluctant to walk (known as being "ambulatory"), preferring to stay in a crouched posture and possibly may have a base-wide stance

CAUSES

- Unknown
- Previous upper respiratory tract infection has been suspected in some patients; relationship not confirmed

RISK FACTORS

- Reports of increased number of cases in the summer and early fall, possibly after outbreaks of upper respiratory disease (not proven); disease can occur throughout the year



TREATMENT

HEALTH CARE

- Usually outpatient
- Inpatient—severely affected patient may require a short period of hospitalization for supportive care
- Treatment is supportive only
- Severe disease—may require initial intravenous or subcutaneous fluids; maintain patient in quiet, well-padded cage initially

ACTIVITY

- Restricted, according to the degree of disorientation and wobbly, incoordinated or "drunken" appearing gait or movement (ataxia)

DIET

- No specific changes or restrictions required
- Patient initially may be reluctant to eat and drink because of disorientation or nausea



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Sedatives—for severe disorientation and rolling; examples are diazepam and acepromazine
- Medications to control nausea and vomiting (known as “antiemetic drugs”) and drugs against motion sickness—usually ineffective; example is meclizine
- Steroids—not recommended; steroids do not alter the course of the disease
- Antibiotics—have been recommended when infection/inflammation of the middle ear (known as “otitis media”) and inner ear (known as “otitis interna”) cannot be ruled out; examples are trimethoprim-sulfa, a first-generation cephalosporin (such as cephalexin), and amoxicillin/clavulanic acid



FOLLOW-UP CARE

PATIENT MONITORING

- Nervous system examination—repeat in approximately 72 hours to confirm stabilization and initial improvement
- Discharge inpatient when able to walk (ambulate), eat and drink

POSSIBLE COMPLICATIONS

- Uncommon
- Dehydration and electrolyte imbalance (rare)

EXPECTED COURSE AND PROGNOSIS

- Marked improvement (especially the irregular eye movements [nystagmus]) within 72 hours, with progressive improvement of the gait and head tilt
- Patients usually normal within 2 to 3 weeks
- Head tilt—final sign to resolve; mild residual tilt may remain
- If signs do not improve rapidly, other causes of vestibular disease should be evaluated
- Rarely recurs; mild head tilt and wobbly, incoordinated or “drunken” appearing gait or movement (ataxia) may return temporarily with stress (such as following general anesthesia)



KEY POINTS

- Despite the initial alarming and incapacitating signs, the prognosis for rapid and complete recovery is excellent

VESTIBULAR DISEASE IN SENIOR DOGS



BASICS

OVERVIEW

- Sudden (acute) nonprogressive disturbance of the peripheral vestibular system in senior dogs
- The vestibular system controls the animal's sense of equilibrium, balance, and orientation; it is composed of the inner ear, nerves, and brain

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- None reported
- Seems to occur more frequently in medium-to-large breeds

Mean Age and Range

- Senior dogs; pets usually greater than 8 years of age

SIGNS/OBSERVED CHANGES in the ANIMAL

- Sudden onset of imbalance, disorientation, reluctance to stand, and (usually) head tilt and irregular eye movements (known as “nystagmus”)
- May be preceded or accompanied by nausea and vomiting
- Head tilt—mild to marked; occasionally erratic side-to-side head movements
- Mild to marked disorientation and wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”) with tendency to lean or fall in the direction of the head tilt
- Strength is normal
- May have base-wide stance

CAUSES

- Unknown



TREATMENT

HEALTH CARE

- Usually outpatient
- Severe disease—patients that cannot walk (known as being “nonambulatory”) or require intravenous fluid support should be hospitalized during the initial stages
- Treatment is supportive, including rehydration and/or maintenance intravenous fluids, if necessary
- Keep recumbent patients warm and dry using soft, absorbent bedding
- Severe disease—physical therapy, including passive manipulation of limbs and moving body to alternate sides, may be required initially

ACTIVITY

- Restrict activity as required by the degree of disorientation and wobbly, incoordinated or “drunken” appearing gait or movement (ataxia)

DIET

- Usually no modification required
- Nausea, vomiting, and severe disorientation—initially withhold food intake by mouth



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Sedatives—for severe disorientation and wobbly, incoordinated or “drunken” appearing gait or movement (ataxia), such as diazepam
- Medications to control nausea and vomiting (known as “antiemetic drugs”) or drugs against motion sickness—questionable benefit; medications include dimenhydrinate and meclizine
- Steroids—not recommended, especially in senior patients that may have low fluid intake; steroids do not alter the course of the disease
- Antibiotics—advised when infection/inflammation of the middle ear (known as “otitis media”) and inner ear (known as “otitis interna”) cannot be ruled out; examples are trimethoprim-sulfa, first-generation cephalosporin (such as cephalexin), and amoxicillin/clavulanic acid



FOLLOW-UP CARE

PATIENT MONITORING

- Nervous system examination—repeat in 2 to 3 days, to confirm stabilization and initial improvement
- Discharge inpatient when able to walk (known as being “ambulatory”), eat and drink

POSSIBLE COMPLICATIONS

- Fluid and electrolyte imbalances and inability to offset kidney insufficiency (if pet has decreased kidney function)—may follow vomiting and/or insufficient fluid and food intake

EXPECTED COURSE AND PROGNOSIS

- Improvement of clinical signs usually starts within 72 hours, with resolution of vomiting and improvement of irregular eye movements (nystagmus) and wobbly, incoordinated or “drunken” appearing gait or movement (ataxia)
- Head tilt and wobbly, incoordinated or “drunken” appearing gait or movement (ataxia)—significant improvement usually occurs over 7 to 10 days; if no improvement in this time, other causes of vestibular disease should be evaluated
- Mild head tilt may remain
- Most patients return to normal within 2 to 3 weeks
- Recurrence—rare; brief return of signs may occur with stress (such as following anesthesia); repeat episodes of vestibular disease in dogs can occur on the same or opposite side, but are uncommon



KEY POINTS

- Although the initial signs can be alarming and incapacitating, the prognosis for rapid improvement and recovery is excellent

SUDDEN (ACUTE) VOMITING



BASICS

OVERVIEW

- Forceful ejection of stomach contents up through the mouth
- “Acute” is an adjective used in medical writing to indicate a sudden or rapid onset and short course of a disease or medical condition
- Sudden (acute) vomiting is defined as vomiting of short duration (less than 5 to 7 days) and of variable frequency
- The gastrointestinal tract includes the stomach, small intestines, and large intestines (known as the “colon”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Variable vomiting of food and/or fluid (may be clear, yellow-tinged [containing bile from the upper small intestine], or blood-stained)
- Ingestion of foreign material
- Variable sluggishness (lethargy) and appetite loss; may see diarrhea and/or black, tarry stools (due to the presence of digested blood; condition known as “melena”)
- May include signs of dehydration, such as dry gums and normally moist tissues of the body (moist tissues are known as “mucous membranes”); reduced skin turgor (turgor is the normal fullness or tension of tissues resulting from fluid content); sunken eyes; pale mucous membranes; rapid heart rate (known as “tachycardia”); and weak pulses; other findings on physical examination may include fluid-filled bowel loops; excessive gut sounds; abdominal pain, which may be localized (such as from a foreign body; inflammation of the pancreas [known as “pancreatitis”]; inflammation/infection of the kidneys [known as “pyelonephritis”]; and liver disease) or may be generalized or diffuse (such as from inflammation of the lining of the abdomen [known as “peritonitis”] or severe inflammation of the intestines [known as “enteritis”]); or an abdominal mass (such as a foreign body; folding of one segment of the intestine into another segment [known as “intussusception”]; twisted abdominal organs)
- May see fever with infectious and inflammatory causes

CAUSES

- Adverse food reactions—indiscretions (eating rapidly, ingestion of foreign material); intolerances (such as sudden diet change, allergies)
- Drugs—antibiotics, anti-inflammatory drugs (such as steroids and non-steroidal anti-inflammatory drugs [NSAIDs]); chemotherapeutic drugs; heart medication (such as digitalis); narcotics; xylazine, a sedative; drug to treat heartworm disease (thiacetarsamide)
- Inflammation of the gastrointestinal tract—infectious inflammation of the intestines (enteritis): viruses (canine parvovirus, canine distemper virus, canine corona virus, feline parvovirus [panleukopenia]); bacteria (*Salmonella*, *Campylobacter*); very sudden (known as “peracute”) bloody inflammation of the intestines (known as “hemorrhagic enteritis”) of dogs
- Ulcers of the stomach or upper small intestine (known as the “duodenum”)
- Blockage or obstruction of the gastrointestinal tract—such as caused by foreign bodies; folding of one segment of the intestine into another segment (intussusception); cancer; stomach dilating with gas and/or fluid (known as “gastric dilatation”), and subsequently rotating around its short axis (known as “volvulus”)—condition known as “gastric dilatation-volvulus” or “bloat;” constipation
- Generalized (systemic) disease—excess levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”); liver failure; sepsis (presence of pus-forming bacteria and their poisons in the blood or tissues); increased levels of acid in the body (known as “acidosis”); electrolyte imbalance (such as low levels of potassium in the blood [known as “hypokalemia”]; low levels of calcium in the blood [known as “hypocalcemia”]; and high levels of calcium in the blood [known as “hypercalcemia”])
- Abdominal disorders—inflammation of the pancreas (pancreatitis); inflammation of the lining of the abdomen (peritonitis); and inflammation with accumulation of pus in the uterus (known as “pyometra”)
- Endocrine disease—inadequate production of steroids by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”); condition in which levels of acid are increased in the blood due to the presence of ketone bodies secondary to diabetes (known as “diabetic ketoacidosis”)
- Nervous system disease—vestibular disturbances (inner ear problems leading to “dizziness” and nausea); inflammation of the membranes covering the brain and spinal cord (known as “meningitis”); inflammation of the brain (known as “encephalitis”); central nervous system trauma
- Parasitism—roundworms (ascarids), *Giardia*, *Physaloptera*, *Ollulanus tricuspis* (cats), salmon poisoning (dogs), *Helicobacter*
- Toxins—lead, ethylene glycol, zinc, fungal toxins (known as “mycotoxins”), household plants

- Miscellaneous—anaphylaxis, heat stroke, motion sickness, pain, fear



TREATMENT

HEALTH CARE

- The most frequent cause of sudden (acute) vomiting is dietary indiscretion (that is, eating something that should not be eaten or eating something that is different from the normal diet)
- Patients with non-serious vomiting are treated on an outpatient basis, resting the gastrointestinal tract by keeping the animal off food and water (known as “NPO” or “nothing by mouth”) for 12 to 24 hours
- If vomiting resolves, initially offer small amounts of water or ice cubes and if vomiting does not recur, follow with an easily digestible, low-fat, single-protein and single-carbohydrate source diet (such as non-fat cottage cheese or skinless white chicken and rice at a 1:3 ratio)
- If vomiting does not recur, wean the patient back onto the normal diet over 4 to 5 days
- Patients with serious vomiting should be hospitalized, and treated initially by withholding food and water (NPO) and providing intravenous (IV) fluids, while further diagnostics are performed

DIET

- Withholding food and water (NPO) for 12 to 24 hours, followed by a bland diet usually will control non-serious vomiting

SURGERY

- Surgery may be indicated, based on the underlying cause of the vomiting (for example, gastrointestinal foreign body)



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Drugs to control nausea and vomiting (known as “antiemetics”) can be used for frequent vomiting
- May use drugs to control nausea and vomiting (antiemetics) in patients with severe vomiting causing electrolyte and/or acid–base disturbances or inflammation caused by reverse flow of stomach contents into the esophagus (known as “reflux esophagitis”)
- Several drugs to control nausea and vomiting (antiemetics) are available for both dogs and cats—phenothiazine derivatives (such as chlorpromazine) and metoclopramide; H₁-receptor antagonists (such as diphenhydramine) can be used in motion sickness for dogs only
- Ulcers of the stomach and/or upper small intestine—can use H₂-blockers (such as ranitidine, which also increases stomach emptying) and/or the stomach lining protectant (sucralfate)
- Antibiotics (such as ampicillin or metronidazole) may be indicated in cases with fever or evidence of stomach/upper intestine lining injury (such as vomiting blood [known as “hematemesis”] or black, tarry stools [due to the presence of digested blood; condition is melena])
- H₂-blockers (such as cimetidine, famotidine, nizatidine)
- Drugs that improve the propulsion of contents through the stomach and intestines (known as “gastrointestinal prokinetic agents”), such as cisapride



FOLLOW-UP CARE

PATIENT MONITORING

- If frequency of vomiting increases or serious problems occur, hospitalize animals for treatment and obtain appropriate diagnostics
- If vomiting persists beyond 7 days, despite medical treatment, pursue appropriate testing for long-term (chronic) vomiting

PREVENTIONS AND AVOIDANCE

- Maintain pet on a consistent diet; do not change food abruptly

- Keep pet out of trash and monitor pet when outside to avoid eating inappropriate materials (such as rocks, bones)

POSSIBLE COMPLICATIONS

- Aspiration pneumonia
- Inflammation of the esophagus (the tube running from the throat to the stomach; condition known as “esophagitis”)

EXPECTED COURSE AND PROGNOSIS

- Recovery from non-serious vomiting is usually rapid and spontaneous



KEY POINTS

- Sudden (acute) vomiting is defined as vomiting of short duration (less than 5 to 7 days) and of variable frequency
- The most frequent cause of sudden (acute) vomiting is dietary indiscretion (that is, eating something that should not be eaten or eating something that is different from the normal diet)
- Maintain pet on a consistent diet; do not change food abruptly
- Keep pet out of trash and monitor pet when outside to avoid eating inappropriate materials (such as rocks, bones)
- Recovery from non-serious vomiting is usually rapid and spontaneous

LONG-TERM (CHRONIC) VOMITING



BASICS

OVERVIEW

- Persistent vomiting lasting longer than 5 to 7 days or vomiting that occurs intermittently several days per week and is not responsive to medical treatment designed to control vomiting (known as “symptomatic treatment”)
- The gastrointestinal tract includes the stomach, small intestines, and large intestines (known as the “colon”)

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

Breed Predispositions

- Confirmed or suspected breed predispositions—Lhasa apsos, shih tzu, and other short-nosed, flat-faced (brachycephalic) breeds are prone to blockage of the pylorus (the muscular area where the stomach empties into the upper small intestine; condition known as “pyloric outflow obstruction”) secondary to enlargement of the lining of the gastrointestinal tract (known as “mucosal hypertrophy”); basenjis, German shepherd dogs, and Chinese shar peis are prone to inflammatory bowel disease (IBD); rottweilers are prone to development of masses or nodular lesions containing a type of white blood cell (called “eosinophils”) in the stomach (condition known as “gastric eosinophilic granulomas”); Airedale terriers are prone to cancer of the pancreas (known as “pancreatic carcinoma”); beagles, Bedlington terriers, cocker spaniels, Doberman pinschers, Labrador retrievers, Skye terriers, and standard poodles are prone to long-term inflammation of the liver (known as “chronic hepatitis”)
- Linear foreign bodies (such as string or yarn) are more common in cats

Mean Age and Range

- Young animals are more likely to ingest foreign bodies

SIGNS/OBSERVED CHANGES in the ANIMAL

- Vomiting of food, clear or yellow-tinged (due to the presence of bile from the upper small intestine) fluid; vomiting blood (known as “hematemesis”); decreased appetite (anorexia); eating of nonfood items (known as “pica”); black, tarry stools (due to the presence of digested blood; condition known as “melena”); increased thirst (known as “polydipsia”); and abdominal distension are typical of diseases involving the stomach
- Diarrhea and profound weight loss are more characteristic of diseases involving the intestines
- Signs such as weakness; increased urination (known as “polyuria”); or yellowish discoloration to the gums and other tissues of the body (known as “jaundice” or “icterus”) relate to other underlying metabolic diseases that may cause long-term (chronic vomiting)
- Weight loss and poor hair coat may indicate long-term (chronic) malnutrition
- Abdominal distention, pain, thickened bowel loops, or masses may be detected during physical examination
- “Tacky” or slightly sticky gums and prolonged skin tenting if dehydration is present; pale membranes if patient has low red-blood cell count (known as “anemia”)

CAUSES

Disease of the Esophagus (the tube running from the throat to the stomach)

- Hiatal hernia
- Backward or reverse flow of stomach contents into the esophagus (known as “gastroesophageal reflux”)
- Inflammation of the lower esophagus (known as “distal esophagitis”)

Infectious Disease

- *Helicobacter*-related inflammation of the stomach (known as “gastritis”)
- Histoplasmosis, a disease caused by *Histoplasma* (a deep fungal infection)
- *Pythium*, a water mold that causes pythiosis
- Small intestinal bacterial overgrowth (SIBO)
- Stomach parasites—*Physaloptera*
- Intestinal parasitism

Metabolic Diseases

- Kidney disease
- Disease of the liver and/or bile system (known as “hepatobiliary disease”)

- Inadequate production of steroids by the adrenal gland (known as “hypoadrenocorticism” or “Addison’s disease”)
- Long-term (chronic) inflammation of the pancreas (pancreatitis)
- Condition in which levels of acid are increased in the blood due to the presence of ketone bodies secondary to diabetes (known as “diabetic ketoacidosis”)
- Metabolic acidosis (a condition in which levels of acid are increased in the blood)
- Electrolyte abnormalities—decreased levels of potassium in the blood (known as “hypokalemia”); increased levels of potassium in the blood (known as “hyperkalemia”); decreased levels of sodium in the blood (known as “hyponatremia”); and increased levels of calcium in the blood (known as “hypercalcemia”)

Inflammatory Bowel Disease (IBD)

- Inflammation characterized by the type of cells present (such as lymphocytic, plasmacytic, or eosinophilic IBD) or the presence of nodular lesions (known as “granulomatous IBD”)
- May involve inflammation of the stomach (gastritis), intestines (enteritis), or colon (known as “colitis”)

Obstructive Gastrointestinal Disease

- Foreign body
- Congenital (present at birth) narrowing of the pylorus (the muscular area where the stomach empties into the upper small intestine; condition known as “pyloric stenosis”)
- Long-term (chronic) enlargement of the tissue of the pylorus (the muscular area where the stomach empties into the upper small intestine; condition known as “pyloric hypertrophic gastropathy”)
- Folding of one segment of the intestine into another segment (known as “intussusception”)

Tumors or Cancer

- Cancer of the gastrointestinal tract: lymphoma, adenocarcinoma, fibrosarcoma
- Cancer of the pancreas (pancreatic adenocarcinoma)
- Tumor of the pancreas that produces gastrin, a hormone that causes the stomach to produce hydrochloric acid (HCl); tumor known as a “gastrinoma”
- Generalized (systemic) mastocytosis (condition in which an abnormal number of mast cells are present in multiple tissues; mast cells contain histamine, and if it is released, it stimulates stomach-acid secretion)
- Nervous system disorders
- Fluid build-up in the brain (known as “cerebral edema”)
- Central nervous system (brain, spinal cord) tumors
- Inflammation of the brain (known as “encephalitis”) or inflammation of the brain and its surrounding membranes (known as “meningoencephalitis”)
- Vestibular disease (inner ear problems leading to “dizziness” and nausea)

Motility Disorders

- Following “gastric dilatation-volvulus” or “bloat” (condition in which the stomach dilates with gas and/or fluid [known as “gastric dilatation”], and subsequently rotates around its short axis [known as “volvulus”])
- Following surgical procedures—such as surgery involving the stomach, pylorus, and upper small intestine (duodenum)
- Electrolyte imbalances

Miscellaneous

- Drug-induced (such as from nonsteroidal anti-inflammatory drugs [NSAIDs], steroids, antibiotics, antifungals)
- Food intolerance/allergy
- Toxicity

Additional Causes in Cats

- Parasitic—heartworm disease (known as “dirofilariasis”), *Ollulanus tricuspis*, *Giardia*
- Inflammatory—inflammation of the gallbladder (known as “cholecystitis”); inflammation of the bile ducts and liver (known as “cholangiohepatitis”)
- Metabolic—increased levels of thyroid hormone (known as “hyperthyroidism”)
- Functional—constipation/obstipation

RISK FACTORS

- Breed-associated disease



TREATMENT

HEALTH CARE

- Specific treatment should be aimed at eliminating the underlying cause in addition to supportive therapy (such as administration of fluids)
- If vomiting persists, stop oral intake of food and water for several days
- Use fluid therapy to replace deficits and to provide for maintenance and ongoing losses of body fluids
- Supplement potassium, if low levels of potassium in the blood (hypokalemia) are present
- Blood transfusion in patients with severely low levels of red-blood cells (severe anemia), with evidence of active bleeding into the gastrointestinal tract

DIET

- Debilitated patients and those in poor nutritional condition may need supplemental feeding by feeding tubes or intravenous feeding
- Dietary therapy for patients with suspected food allergy or with inflammatory bowel disease (IBD) should use a diet containing a single-source protein novel to the patient (that is, feeding a protein to which the animal has never been exposed)

SURGERY

- Use surgical treatment if uncontrolled bleeding, blockage or obstruction, or abnormal opening (known as a “perforation”) of the gastrointestinal tract is identified



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Anti-secretory drugs, such as H₂-blockers (such as cimetidine, ranitidine, famotidine, nizatidine), or proton-pump inhibitors, such as omeprazole (more potent)
- Protectants (such as sucralfate) to accelerate stomach lining healing; can be used with anti-secretory drugs for patients with evidence of upper gastrointestinal bleeding (signs include vomiting blood [hematemesis] or black, tarry stools [melena])
- Antibiotics—indicated for treatment of *Helicobacter*-associated inflammation of the stomach (gastritis); in addition to steroids in the treatment of inflammatory bowel disease (IBD), and to treat small intestinal bacterial overgrowth (SIBO) syndrome
- Suggested treatment of *Helicobacter*-associated inflammation of the stomach (gastritis)—amoxicillin plus omeprazole and metronidazole; clarithromycin can be used with amoxicillin and metronidazole as an alternative therapy for cats
- Metronidazole—may be used in conjunction with steroids to treat inflammatory bowel disease (IBD)
- Small intestinal bacterial overgrowth (SIBO) syndrome—tetracycline, metronidazole, amoxicillin, and tylosin, in addition to correcting the underlying cause
- Use steroids in conjunction with dietary changes and metronidazole to treat biopsy-confirmed inflammatory bowel disease (IBD); azathioprine, chlorambucil, or cyclosporine also can be used in patients with poor response to steroids alone or to decrease the dosage of steroids required to control signs
- Drugs that improve the propulsion of contents through the stomach and intestines (known as “gastrointestinal prokinetic agents,” such as metoclopramide or erythromycin) are used to treat delayed stomach emptying, not associated with obstructive disease
- Pyrantel pamoate is effective for *Physaloptera*; fenbendazole is effective for *Ollulanus*
- Iron supplementation for animals with long-term (chronic) gastrointestinal bleeding that develop blood-loss anemia
- Surgery and/or chemotherapy for cancer, depending on the tumor type and location
- Excessive production of stomach acid, as occurs with mastocytosis and gastrin-secreting pancreatic tumors, is best treated with potent anti-secretory drugs (such as omeprazole) to diminish inflammation of the stomach (gastritis), stomach ulcer, and long-term (chronic) vomiting
- Reserve drugs to control nausea and vomiting (known as “antiemetics”) for patients with persistent vomiting unresponsive to treatment of the underlying disease
- Vomiting caused by chemotherapy is best treated with ondansetron, given 30 minutes before chemotherapy



FOLLOW-UP CARE

POSSIBLE COMPLICATIONS

- Aspiration pneumonia
- Inflammation of the esophagus (the tube running from the throat to the stomach; condition known as “esophagitis”)



KEY POINTS

- Chronic vomiting is persistent vomiting lasting longer than 5 to 7 days or vomiting that occurs intermittently several days per week and is not responsive to medical treatment designed to control vomiting (known as “symptomatic treatment”)
- Specific treatment should be aimed at eliminating the underlying cause in addition to supportive therapy
- Debilitated patients and those in poor nutritional condition may need supplemental feeding by feeding tubes or intravenous feeding

VON WILLEBRAND'S DISEASE

(A BLEEDING DISORDER)



BASICS

OVERVIEW

- Primary bleeding defect caused by low levels of von Willebrand's factor or decreased function of existing von Willebrand's factor; von Willebrand's factor is a type of protein that binds to platelets, causing them to crowd or mass together (aggregate) and to adhere to one another to stop bleeding—if levels of von Willebrand's factor are low or if the existing von Willebrand's factor does not function normally, platelets do not aggregate and adhere to one another and bleeding is not stopped
- "Platelets" and "thrombocytes" are names for the normal cell fragments that originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to "plug" tears in the blood vessels and to stop bleeding
- Clinical expression of von Willebrand's disease varies from mild to severe bleeding

GENETICS

- Most common hereditary bleeding defect seen in dogs
- An autosomal trait in dogs; both males and females express and transmit the defect with equal frequency
- Expression pattern of severe forms (Types 2 and 3 von Willebrand's disease) is recessive; milder form (Type 1 von Willebrand's disease) appears to be recessive or incomplete dominant

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs
- Rarely reported in cats

Breed Predilections

- Three type classifications are found in dogs; a single type predominates within each affected breed:
 - Type 1 von Willebrand's disease (mild to moderate signs): decreased protein (von Willebrand's factor) levels; type 1 is the most common classification; seen in the following breeds: Airedale, Akita, basset hound, Bernese mountain dog, dachshund, Doberman pinscher, German shepherd dog, golden retriever, greyhound, Irish wolfhound, Manchester terrier, miniature pinscher, Pembroke Welsh corgi, and poodle; sporadic cases are seen in any breed and mixed-breed dogs
 - Type 2 von Willebrand's disease (severe signs): decreased protein (von Willebrand's factor) levels and decreased function of existing von Willebrand's factor; seen in the following breeds: German wirehaired pointer and German shorthaired pointer
 - Type 3 von Willebrand's disease (severe signs): complete lack of protein (von Willebrand's factor); seen in the following breeds: Chesapeake Bay retriever, Dutch Kooiker dog, Scottish terrier, and Shetland sheepdog; sporadic cases are seen in any breed

Mean Age and Range

- Severe signs (Types 2 and 3 von Willebrand's disease) typically are apparent by 4 to 6 months of age
- Milder forms typically are characterized by abnormal bleeding after surgery or trauma, or in association with another condition that impairs blood clotting

SIGNS/OBSERVED CHANGES in the ANIMAL

- Bleeding from moist tissues of the body: nose bleed (known as "epistaxis"); bleeding into the gastrointestinal tract (the gastrointestinal tract includes the stomach, small intestines, and large intestines); blood in the urine (known as "hematuria"); bleeding into the vagina; and bleeding gums (known as "gingival hemorrhage")
- Prolonged bleeding after surgery or trauma
- Blood loss-related, low red-blood cell count (known as "blood-loss anemia"), if prolonged bleeding

CAUSES

- Hereditary von Willebrand's disease is caused by mutations that impair production, release, or stability of von Willebrand's factor (type of protein that binds to platelets, causing them to crowd or mass together [aggregate] and to adhere to one another to stop bleeding)

RISK FACTORS

- Acquired (condition that develops sometime later in life/after birth) disease conditions or drug therapy that impair platelet function may worsen clinical signs of von Willebrand's disease; "platelets" and "thrombocytes" are names for the normal cell fragments that

originate in the bone marrow and travel in the blood as it circulates through the body; platelets act to “plug” tears in the blood vessels and to stop bleeding



TREATMENT

HEALTH CARE

- Transfusion of fresh whole blood, fresh plasma, fresh frozen plasma, and cryoprecipitate (a plasma component used to treat bleeding disorders) will supply von Willebrand's factor (type of protein that binds to platelets, causing them to crowd or mass together [aggregate] and to adhere to one another to stop bleeding)
- Component therapy (fresh frozen plasma or cryoprecipitate) is best for surgical prophylaxis and nonanemic patients
- Patients with severe von Willebrand's disease may require repeated transfusion (every 6 to 12 hours) to control or prevent bleeding

ACTIVITY

- Depends on severity of clinical signs

SURGERY

- Pre-operative transfusion should be given just before the surgical procedure
- Cage rest and close monitoring (serial determination of packed cell volume [“PCV,” a means of measuring the percentage volume of red-blood cells as compared to the fluid volume of blood] and examination of surgical site) for 24 hours after surgery are ideal to confirm adequate control of bleeding
- Management of severe von Willebrand's disease typically requires at least 1 postoperative transfusion



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Desmopressin acetate (DDAVP) can be given pre-operatively to dogs with mild to moderate Type 1 von Willebrand's disease to enhance control of bleeding during and after surgery
- Response is variable; transfusion should be available if DDAVP alone does not prevent bleeding



FOLLOW-UP CARE

PATIENT MONITORING

- Observe closely for bleeding associated with trauma or surgical procedures

PREVENTIONS AND AVOIDANCE

- Screen dogs pre-operatively to determine baseline von Willebrand's factor levels in breeds or lines with high prevalence of von Willebrand's disease; the risk of abnormal bleeding is greatest for dogs with von Willebrand's factor levels of less than 25%; von Willebrand's factor is a type of protein that binds to platelets, causing them to crowd or mass together (aggregate) and to adhere to one another to stop bleeding
- Clinically affected dogs should not be bred; carriers of von Willebrand's disease can be identified based on low von Willebrand's factor levels of less than 50%; however, values for carrier and clear dogs may overlap at the low end of normal range (von Willebrand's factor levels of 50% to 70%)
- At present, commercial tests (VetGen™ von Willebrand's DNA test) to detect specific von Willebrand's disease mutations in DNA are available for several breed-variants of von Willebrand's disease; dogs that are heterozygous for a specific mutation are considered von Willebrand's disease “carriers” and homozygotes are considered von Willebrand's disease “affected”
- Selective breeding practices can reduce or eliminate von Willebrand's disease from an affected pedigree—breeding two clear parents is ideal, because all offspring are expected to be von Willebrand's disease clear; breeding one clear and one carrier parent may be acceptable, with the clear puppies produced from this mating used for subsequent breeding
- Carrier to carrier matings are inadvisable, because they most likely will produce clinically affected offspring

POSSIBLE COMPLICATIONS

- Depends on severity of clinical signs
- Bleeding during and after surgery or trauma

EXPECTED COURSE AND PROGNOSIS

- Most dogs with mild to moderate von Willebrand's disease have good quality of life and require minimal or no special treatment
- Dogs with more severe forms require transfusion for surgery, and should be transfused if supportive care fails to control spontaneous bleeding; most of these dogs can be maintained comfortably in pet homes



KEY POINTS

- Most common hereditary bleeding defect seen in dogs
- Most dogs with mild to moderate von Willebrand's disease have good quality of life and require minimal or no special treatment
- Dogs with more severe forms require transfusion for surgery, and should be transfused if supportive care fails to control spontaneous bleeding; most of these dogs can be maintained comfortably in pet homes
- Clinically affected dogs should not be bred
- Selective breeding practices can reduce or eliminate von Willebrand's disease from an affected pedigree

WEIGHT LOSS AND CACHEXIA



BASICS

OVERVIEW

- Weight loss is considered clinically important when it exceeds 10% of the normal body weight and is not associated with fluid loss
- “Cachexia” is defined as a general physical wasting and malnutrition characterized by extreme weight loss, muscle wasting, lack of appetite (known as “anorexia”), and general debilitation that is associated with long-term (chronic) disease or inflammation

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs and cats

SIGNS/OBSERVED CHANGES in the ANIMAL

- Clinical signs of particular diagnostic value in patients with weight loss are whether the appetite is normal, increased, decreased, or absent, and the presence or absence of fever or other signs of generalized (systemic) illness
- Historical information is extremely important—evaluate the type and quantity of diet being offered, the pet’s daily activity, environment, appetite, signs of gastrointestinal disease (such as vomiting, diarrhea, stool color/consistency, difficulty swallowing [known as “dysphagia”])
- Signs associated with an underlying disease

CAUSES

Dietary Causes

- Insufficient quantity of food
- Poor quality of food
- Inedible food—decreased palatability
- Spoiled diets
- Diets that have lost nutrients because of prolonged storage
- Competition in a multi-pet household

Lack of Appetite (Anorexia)

- Pseudoanorexia or “false lack of appetite,” a condition in which the animal does not eat because it has difficulty grasping, chewing, and/or swallowing food
- Inability to smell, grasp, or chew food
- Difficulty swallowing (dysphagia)
- Regurgitation (return of food or other contents from the esophagus or stomach back up through the mouth)
- Vomiting

Disorders in which Nutrients Are Not Absorbed from the Intestines (Malabsorptive Disorders)

- Infiltrative and inflammatory bowel disease
- Lymphangiectasia—dilation of the lymphatic vessels in the gastrointestinal tract; the “gastrointestinal tract” includes the stomach, small intestines, and large intestines
- Severe intestinal parasitism

Disorders in which Nutrients Are Not Digested (Maldigestive Disorders)

- Exocrine pancreatic insufficiency—a syndrome caused by inadequate production and secretion of digestive enzymes by the pancreas

Metabolic Disorders

- Organ failure—heart failure, liver failure, and kidney failure
- Inadequate production of steroids by the adrenal glands (known as “hypoadrenocorticism” or “Addison’s disease”)
- Excessive production of thyroid hormone (known as “hyperthyroidism”), especially in cats
- Extreme weight loss and muscle wasting (cachexia) due to cancer

Excessive Nutrient Loss

- Protein-losing enteropathy—condition in which proteins are lost from the body through the intestines
- Protein-losing nephropathy—condition in which proteins are lost from the body through the kidneys
- Diabetes mellitus (“sugar diabetes”)

- Extensive skin lesions (such as burns)

Disease of the Nervous System and Muscles

- Disease of the nerves that connect the spinal cord and muscles (known as “lower motor neuron disease”)
- Central nervous system (brain, spinal cord) disease—usually associated with lack of appetite (anorexia) or the animal does not eat because it has difficulty grasping, chewing, and/or swallowing food (pseudoanorexia)

Excessive Use of Calories

- Increased physical activity
- Prolonged or extreme cold environment
- Excessive levels of thyroid hormone (hyperthyroidism)
- Pregnancy or lactation
- Increased breakdown of lean muscle mass and body tissues (known as “catabolism”)—fever, infection, inflammation, cancer



TREATMENT

HEALTH CARE

- The most important treatment principle is to treat the underlying cause of the weight loss
- Determine caloric requirements for the animal, taking into account activity level and severity of illness

DIET

- Must provide sufficient caloric nutrition in the form of adequate amounts of an appropriate, high-quality diet—fed in the form or manner that best allows patient utilization
- Patient may need to be fed by feeding tube or through intravenous feeding (known as “parenteral nutrition”)

SURGERY

- Placement of a feeding tube may be necessary



MEDICATIONS

- Depend on the underlying cause of the weight loss



FOLLOW-UP CARE

PATIENT MONITORING

- Depends on the underlying cause of the weight loss
- Patient should be weighed regularly and frequently
- Appetite and caloric intake should be monitored closely to ensure nutritional needs are being met

PREVENTIONS AND AVOIDANCE

- Depend on the underlying cause of the weight loss

POSSIBLE COMPLICATIONS

- Depend on the underlying cause of the weight loss

EXPECTED COURSE AND PROGNOSIS

- Depend on the underlying cause of the weight loss



KEY POINTS

- Weight loss is considered clinically important when it exceeds 10% of the normal body weight and is not associated with fluid loss
- “Cachexia” is defined as a general physical wasting and malnutrition characterized by extreme weight loss, muscle wasting, lack of appetite (known as “anorexia”), and general debilitation that is associated with long-term (chronic) disease or inflammation
- Clinical signs of particular diagnostic value in patients with weight loss are whether the appetite is normal, increased, decreased, or absent, and the presence or absence of fever or other signs of generalized (systemic) illness

WOBBLER SYNDROME (CERVICAL SPONDYLOMYELOPATHY)



BASICS

OVERVIEW

- “Wobbler syndrome” (also known as “cervical spondylomyelopathy”) is a disease of the neck (cervical spine), commonly seen in large- and giant-breed dogs
- Wobbler syndrome is characterized by compression of the spinal cord and/or nerve roots, which leads to nervous system deficits and/or neck pain
- The spine is composed of multiple bones with disks (intervertebral disks) located in between adjacent bones (vertebrae); the disks act as shock absorbers and allow movement of the spine; the vertebrae are named according to their location—cervical vertebrae are located in the neck and are numbered as cervical vertebrae one through seven or C₁-C₇

GENETICS

- Genetic basis proposed for the borzoi and basset hound
- No definitive data regarding inheritance of wobbler syndrome in Doberman pinschers

SIGNALMENT/DESCRIPTION of ANIMAL

Species

- Dogs

Breed Predispositions

- Doberman pinschers are affected most commonly, with at least 50% of the cases seen in this breed
- Other breeds with a high incidence of wobbler syndrome include the Great Dane, rottweiler, Weimaraner and Dalmatian
- Wobbler syndrome may be seen in any canine breed, including small-breed dogs

Mean Age and Range

- Doberman pinschers and other large-breed dogs usually are presented to the veterinarian for clinical signs when they are over 3 years of age, with a mean age of 6 years
- Giant-breed dogs usually are presented when they are less than 3 years of age, although signs can develop later in life

Predominant Sex

- Males are slightly more likely to have wobbler syndrome than are females

SIGNS/OBSERVED CHANGES in the ANIMAL

- The classic clinical presentation is a slowly progressive, wobbly, incoordinated or “drunken” appearing gait or movement (known as “ataxia”) of the rear legs, with less severe involvement of the front legs
- Long-term (chronic), slowly progressive abnormal gait
- Sudden (acute) neck pain
- Front leg gait can appear to be shortened, with a floating appearance, or very weak
- Dogs may be unable to walk (known as being “non-ambulatory”)
- Loss of muscle mass of the shoulder (known as “supraspinatus muscle atrophy”) and worn toenails can be seen in some cases

CAUSES

- Nutrition—excess protein, calcium and caloric intake were proposed as causes in Great Danes

RISK FACTORS

- Body conformation—large head and long neck have been proposed, but later studies found no correlation between body dimensions and wobbler syndrome
- Fast growth rate has been proposed, but not confirmed



TREATMENT

HEALTH CARE

- Inpatient, if surgical treatment is elected
- Outpatient, if medical management is chosen as the treatment
- Dogs that cannot walk (non-ambulatory dogs)—keep patients on soft bedding and turn every 4 hours to avoid “bed sores” (known as “decubital ulcers”); empty the bladder on a routine schedule; physiotherapy is essential to avoid loss of muscle mass (muscle atrophy) and stiffening of the joints (known as “ankylosis”), and to hasten recovery

ACTIVITY

- Medically treated dogs should have restricted activity for at least 2 months
- Restriction of activity is important for the first 2 or 3 months following surgery to allow fusing of the backbones (vertebrae) at the site of surgery

DIET

- Avoid excess protein, calcium or caloric intake in giant-breed dogs

SURGERY

- Various surgical procedures have been performed in treating wobblers syndrome
- Recurrence rate is approximately 20% with any surgical technique



MEDICATIONS

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive.

- Steroids—dexamethasone initially; followed by a gradually decreasing dose of prednisone, as directed by your pet’s veterinarian
- Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used in dogs with only increased sensitivity to touch involving the neck (known as “cervical hyperesthesia”) or a slight wobbly, incoordinated or “drunken” appearing gait or movement (ataxia)



FOLLOW-UP CARE

PATIENT MONITORING

- Repeat the nervous system evaluation as often as needed to monitor response to treatment

PREVENTIONS AND AVOIDANCE

- Excessive activity, jumping, running should be avoided
- Avoid use of collars placed around the neck; use a body harness

POSSIBLE COMPLICATIONS

- Seizures and transient nervous system deterioration can occur after special X-ray techniques in which a dye is injected into the spinal canal (procedure known as “myelography”) to allow visualization of the spinal cord
- Recurrence of clinical signs can occur in dogs treated medically or surgically

EXPECTED COURSE AND PROGNOSIS

- 80% of patients improve with surgery
- Approximately 50% patients improve with medical treatment (restricted activity with or without steroids) and 25% remain stable



KEY POINTS

- Surgery offers the best chance of improvement (80%), but a 1% to 5% risk of significant complications is associated with surgical procedures of the neck (cervical spine)